CARDIOVASCULAR SOUND

IN HEALTH AND DISEASE

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Being a Comprehensive Treatise Introduced by a Historical Survey Illustrated Mainly by Sound Spectrograms (Spectral Phonocardiograms) and Supplemented by an Extensive Bibliography

With a Section on Respiratory Sound

By

VICTOR A. McKUSICK, M.D.

Associate Professor of Medicine

The Johns Hopkins University School of Medicine

Physician

The Johns Hopkins Hospital



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Dedicated to the memory of

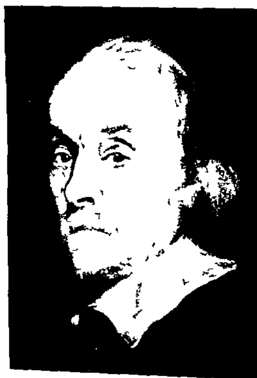
WILLIAM HARVEY

(1578–1657)

whose reference to the heart sounds is the

earliest and clearest which survives

in the Tercentennial Year after his death



WILLIAM HARVEY (1578-1657)
Detail Royal College of Physicians portrait (789)

Preface

Any virtue which this monograph may have will probably be related to one or all of the following features (1) the comprehensive and I trust critical historical survey (2) the use of spectral phonocardiogram for purposes of illustration (3) discussion of auscultatory signs on the basis of physical and physiological principles whenever possible in the present state of knowledge (4) an obsession with numbers graphed relationships and quantification in this field which is still largely qualitatively descriptive and finally (5) the provision of an extensive bibliography

Cardiovascular sound and cardiac auscultation are at the hub of clinical cardiology and cardiovascular physiology The findings of so many other diagnostic methods have pertinence in connection with cardiovascular sound and vice versa and in so few cardiologic conditions is cardiovascular sound of no significance that *an exhaustive* treatise on cardiovascular sound approaches being a textbook of cardiology For this no apology is offered

Why cultivate the art of cardiac auscultation *why study* the science of cardiovascular sound in these days of *cardiac catheterization* and angiocardiography? *In general the reason* is that cardiovascular sound as detected by auscultation contains information on *cardiovascular* function obtainable by *no other method* Levine (887) suggested that the usefulness is *mainly* of three types (1) To exclude certain *diagnoses*, (2) to establish certain diagnoses (3) to provide *clues* to diagnostic possibilities requiring exploration Sometimes it is as *important to exclude* a diagnosis as to establish it *Although rare* cases of murmurless *bacterial endocarditis may* occur auscultation usually make it possible to exclude *this* diagnosis with relative certainty in cases of fever of obscure cause Although there are simulating conditions which may deceive the unwary and unschooled ear the diagnosis of pericarditis aortic regurgitation and mitral stenosis can often be made only by the characteristic auscultatory change Auscultation may provide tips to diagnoses which can be established by other methods e.g. a gallop may suggest *that epigastric pain is of cardiac origin*

Spectral phonocardiographs appear to have *teaching* as well as scientific value In the spectral phonocardiogram the display of the frequency spectrum (its unique feature) is responsible for three advantages of the method (1) Quality, or timbre is given physical definition (2) resolution in the time dimension is improved, (3) a more accurate display of the wide dynamic (i e intensity) range of cardiovascular sound is attained Potentially the method can do all the ear can and probably can even surpass the ear because (1) it is not wed to a particular frequency intensity response *curve* (2) *it suffers* from no psychoacoustic *impediments* (3) *it provides* better resolution in the *time dimension* and (4) it produces permanent quantifiable record

The order of presentation that has been adopted is evident from the Table of Contents A progression from the general to the specific has been practiced General and basic considerations *are first* dealt with then the individual ele*ments of* cardiovascular sound—transients and murmurs *of* various types—have been described finally a long synthesizing section discusses the sonic phenomena which accompany various categories of cardiovascular disease—valvular congenital hypertensive myocardial, pericardial miscellaneous The *index* has been prepared with particular care

Through a happy coincidence, the year 1957, which was spent in the preparation of this monograph, was also the tercentenary of the death of William Harvey, whose early contribution to the field of cardiovascular sound is described on page 3 It is a pleasure, in dedicating to Harvey this survey of the development of the field in the last 300 years, to make a small private addition to the various public observances with which cardiologists and cardiovascular physiologists have taken note of the year

VICTOR A McKUSICK, M D
The Johns Hopkins Hospital

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Dr E V Newman guided our initial exploration of sound spectrography in the medical application Dr S A Talbot has given unstintingly of his time and advice it was he who suggested the designation spectral phonocardiography to indicate that the method is a variety of phonocardiography differing from the conventional oscillographic method mainly in the fact that frequency spectrum is displayed To Mr George N Webb electronic engineer is due major credit for the supervision of instrumental design and operation and for important suggestions in the planning of our studies Mr H Kenneth Wiskind Ph D candidate in the Department of Aeronautics Johns Hopkins University studying problems of sound in fluid systems has interjected a stimulating point of view To five classes of medical students in the Johns Hopkins University School of Medicine guinea pigs for testing the pedagogic value of spectral phonocardiography the writer expresses his appreciation for their forbearance and helpful comment Twenty two medical students have during summers and other free periods engaged productively in the activities of the laboratory Special acknowledgment is due them James I Bradshaw J O'Neal Humphries James A Reid Genevieve E Murray Fernando G Vescia Oliver N Massengale Jr Donald P Hahn George W Santos Robert C Keeler W Paul Reagan Milton Wigod (Medical College of Virginia) Edmund W Kline Mary Betty Stevens Thomas I Y Chen George H Lobley John A Tucker Robert Osborne (M C V) Allen O Warner, Allen L Pusch Gordon K Norwood W Robert Wiman Daniel I Glick Dr Humphries also collaborated during a year's tenure of a research fellowship Dr Samuel Huntington Boyer IV my first research fellow contributed in many ways both to the heart sound studies and to this monograph During the tenure of a fellowship in this laboratory, Dr Arthur W Chisholm of Toronto conducted several studies referred to here and reviewed the manuscript constructively Dr Claude R Garfield also assisted as a research fellow

Miss Eugenia Morgan did a large share of the technical work associated with the study

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SECTION I

Historical Survey

CHAPTLR 1
The History of Cardiovascular Sound[1]

HARVEY TO LAENNEC

It is surprising that no definite reference to the heart sounds dates from the period before William Harvey The Hippocratic writings (687a f) of about 400 B C indicate that the ear was applied to the chest as an established practice in ancient medicine and was successful in demonstrating what is now referred to as the Hippocratic succussion splash of hydropneumothorax Other types of medical sound were described rales (687g h) pleural friction rub (a creak like new leather) (687i j) asthmatic wheezing (212) borborygmi (687) Although they must have been heard the heart sounds were never commented on

One might think the sound produced by the normally beating heart was such a common place personal experience that there was contempt for detailed description and analysis However the derision which greeted description of the heart sound by Harvey and of the fetal heart sound by Marsac (p 4) suggests that existence of the heart sounds was not as common knowledge as one might presume

It would be supposed that at least abnormal varieties of cardiovascular sound particularly loud murmur audible at a distance from the patient would have excited wonder If they did no record survive One can only speculate on how often in primitive and even relatively sophisticated societies the loud raucous murmur of calcific aortic stenosis or of retroverted aortic cusp was interpreted as the cries of an evil spirit inhabiting the patient and causing his symptoms

In 1616 the year that Shakespeare and Cervantes died William Harvey (1578–1657) delivered his visceral lectures According to his note which, written in his own hand were discovered in the British Museum Harvey compared the heart to two clacks of a water bellows to raise water (651) He was probably referring to the two sets of heart valves since *clack* was a word used for *valve* in Harvey s time For example the Oxford Dictionary quotes Bate as writing in 1634 "a clacke is a piece of Leather nayled over any hole having a piece of Lead to make it be close so that the ayre or water in any vessel may thereby bee kept from going out Harvey s use of an onomatopoetic term *clack* may suggest that he then had knowledge of the heart sounds as well as a suspicion of their valvular origin

In his famous *De Motu Cordis* (1628) Harvey wrote (650) as follows with each movement of the heart when there is the delivery of a quantity of blood from the veins to the arteries a pulse take place and can be heard within the chest

There can be no doubt that Harvey was referring to an acoustic phenomenon for a contemporary, Aemilius Parisanus, writing in 1647 stated sarcastically (1180) Nor we poor deafs nor any other doctor in Venice can hear them, but happy is he who can hear them in London Daremberg (1816–1872) a competent French medical historian termed this statement a shameful testament of stupidity (330)

Another clear pre Laennec reference to the heart sounds is that of Robert Hooke (1635–1703), versatile scientist secretary of the Royal Society of London originator of Hooke s law of elasticity

I have been able to hear very plainly the beating of a Man s Heart Who knows I say but that it may be possible to discover the Motions of the Internal

[1] Several general sources (6 a aS 1300b 156 1565) were used in the preparation of this section

Parts of Bodies by the sound they make, that one may discover the Works performed in the several Offices and Shops of a Man's Body, and thereby discover what Instrument or Engine is out of order (710)

In this statement Hooke left no doubt of his familiarity with the heart sounds and in addition foresaw the usefulness of clinical auscultation (Fig 1)

The fetal heart sounds appear to have been known at least as early as 1650 About that year a physician Phillipe Le Goust (869), of Niort, France, wrote a poem making fun of his colleague Marsac for claiming to hear the heart of the fetus "beating like the clapper of a mill" (615) The poem of Le Goust, written partly in Latin, partly in the Limousin dialect, was republished by Phelippeaux in modern French in 1879 (1206) Just as Parisanus by his asinine criticism of Harvey makes it clear that Harvey heard the heart sounds and that the phenomenon was not well known, Le Goust's equally ignorant satire leaves no doubt that Marsac discovered obstetrical auscultation

A number of references to murmurs can be

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Fig 1 Title page of the posthumous publication containing Hooke's reference to the heart sounds

found in writings between Harvey and Laennec. Some were the loud variety *audible at a distance*, others required application of *the ear to the chest* or other part. The following instances represent a sampling.

In 1654 Caspar Bartholin the Elder (1616–1680) wrote (1360) about "a young lady of *Copenhagen* . . . sometimes vexed by a continuous headache. At these times she felt the pulse in the carotid arteries so violently in her head that a sound might be heard from a long distance like a clock."

Another early reference is that of Cornelius Stalpert vander Wiel who was born in 1620 and first published his book in 1687 (1433). Morgagni (1114) quotes him as follows: "For he tells us of a girl being cured by him who had her face pallid and very much swelled, and had never undergone any menstrual purgation, in whom he could very distinctly hear the agitation of the water itself in the pericardium, when the heart was pulsating (for she laboured under a palpitation of the heart)." This patient's disorder was interpreted as hydropneumopericardium by Bricheteau (174) writing in 1844. However, it may be that she had chronic nephritis with uremia and that it was an ordinary, albeit very loud, pericardial friction rub which was *heard*.

In 1715 James Douglas (1676–1742) of St. Bartholomew's Hospital, London, made the *following* observation in a patient found at autopsy to have aortic regurgitation (368): "*Which* is most incredible, *at sometimes* the trembling and the throbbing made such a *noise* in his *Breast* as plainly could be heard *at some* Distance from his Bedside."

Bouillaud (139) states that "*Nysten* long before Laennec discovered auscultation pointed out the more or less gurgling sound which is produced by the *injection* of gas into the cavities of the heart of animals." *Pierre Hubert Nysten* (1771–1818), a physician born in Belgium, spent most of his career in Paris.

In 1757 the noted William Hunter (1718–1783) of London described in a *case of arterio*venous fistula ("varicose aneurism") "a thrill ('particular vibratory movement') and a murmur ('bruissement') which he compared to that produced by air in passing through a small opening, or to the sound of the letter R spoken in a prolonged whisper" (731).

In 1803 Benjamin Travers (1783–1858), then a demonstrator in anatomy at Guy's Hospital, London (1010), and surgeon to the East India Company, reported (1475) on an "aneurism by anastomosis" clearly a carotico-cavernous fistula. "The patient had felt a sudden snap on the left side of her forehead. Thereafter she had a constant noise in her head which to her sensation exactly resembled the blowing of a pair of bellows. The murmur increased when she lowered her head. Protrusion of the left eyeball developed as well as a thrilling tumor at the inner canthus." Travers noted that when he occluded the left carotid the exophthalmos diminished and the "sibilant sound between a hiss and a buzz likewise disappeared." He ligated the left carotid artery with cure of the patient.

Writing on "aneurism" in 1812 (1274) Baron Anthelme Balthasar Richerand (1779–1840) of Paris described a conscript seen in 1803 who sought exemption on the basis of an injury to an arm. "An arteriovenous fistula was found. The ear applied to the bend of the elbow heard a faint murmur produced by the friction of the blood against the margins of the opening between the vein and the artery."

About 1817 Baron Larrey (1766–1842) who during the Napoleonic Wars attained fame by his contributions to military medicine described the murmur of an arteriovenous fistula which had resulted from a saber wound. "The clavicle was obscured by a considerable tumor which had a pulsation synchronous with the pulse. Furthermore one felt and heard more deeply *and in the* direction of the axillary vein a singular murmur ('bruissement') like that which *would be* produced by a liquid which was made *to pass* through many crooked metallic *pipes*" (842).

In 1815 in his *Treatise on Diseases of Arteries* (695) Joseph Hodgson (1788–1869) of Birmingham described a *case of femoral arterio*venous fistula: "*The tumor* . . . presented a particular vibratory movement which caused a certain humming to be heard or rather a hissing corresponding *to the* arterial pulsation . . . The bruit was more distinct *when the* ear was placed on the tumor at the point where the sword had

penetrated It was also readily audible 3 or 4 inches from this point "

Cardiac murmurs were less frequently noted, however In 1806, in his famous *Essai sur les Maladies du Coeur,* Jean Nicholas Corvisart (1755–1821), physician to Napoleon and one of Laennec's teachers, wrote as follows about mitral stenosis (297) "Among the several specific signs which permit recognition of this affection is a particular murmur ('bruissement'), difficult to describe, which shows that the blood is passing through an orifice which is not large enough in proportion to the quantity of fluid to which it must give passage" The word "bruissement" used by pre Laennec French writers for 'murmur' was also used for the "buzzing' (of bees), "rustling" (of leaves), murmuring" (of brook) "rumbling," and so on Corvisart also wrote as follows (298) Some authors assert that they have been able to hear in certain cardiac maladies the sound produced by the beating of this viscus even at a great distance from the bed of the patient I have never had the occasion to verify these observations I have only heard the beatings when the ear was applied to the chest of the patient" However in 1826 Laennec (831*a*) stated The professor never put his ear to the chest

The clearest descriptions of heart murmurs before Laennec are those of Allan Burns (1781–1813) of Glasgow (Fig 2) who in his brief lifetime wrote what is essentially the first textbook



Fig 2 Silhouette Allan Burns (Courtesy of the Library University of Glasgow)



Fig 3 René Théophile Hyacinthe Laennec (1781–1826)

of cardiology in the English language It is true that the murmurs he recorded were not of his own observation He quoted (199 p 187) a Dr Brown and a Dr Rutherford who had a patient with a hissing noise as of several currents meeting the sound was frequently audible as in the varicose aneurism On dissection the mitral valve was found indurated and reticulated There seemed to be an opening left between the auricle and ventricle during the contraction of the latter Thus on each contraction the blood flowed in part into the aorta in part into the left auricle producing regurgitation along the pulmonary veins The regurgitation of the blood from the ventricle into the auricle must have produced the hissing Burns also quotes (199 p 95) the description by a Dr Linton of a patient in whom the violence of the palpitation was such that it was both audible and visible at a distance

Laennec (Fig 3) had made a consistent practice of direct auscultation for several years before he devised the stethoscope He wrote (831*a*) Bayle [Gaspard Laurent B 1774–1816 writer of a monograph on pulmonary tuberculosis] was the first whom I saw employ it when we followed the service of Corvisart together It was obviously from considerable experience that Laennec (831*b*) wrote so feelingly about direct auscultation As inconvenient for the physician as for the patient distaste alone renders it almost impracticable in the hospital it cannot even be

proposed to most women and in most of them the volume of the breast is a physical obstacle to its use. Bayle died the very year that the idea of the stethoscope came to Laennec.

I. J. Double of Paris, in 1817 (366) makes reference to immediate auscultation and seems to have claimed for himself its application to the study of respiratory sound: "We must apply the ear closely to every point of all its [the chest's] aspects, by which means we can distinguish not merely the kind and degree of the sound but even its precise site. . . . I have frequently derived great benefit from this mode of investigation, which is peculiar to myself and to which I was naturally led by the employment of the like method in exploring the pulsation of the heart." In 1831 (404) the British medical press had an amusing account: "M. Double, whose name must be familiar to our readers as that of an eminent physician in Paris, had lately occasion to read a memoir to the Academy of Sciences in which he mentions the following circumstances as having first directed his attention to the sound of the heart. Many years ago, when he was taking leave of his mother, he laid his head upon her bosom and wept in an agony of maternal grief at parting with him, but her philosophic son was otherwise employed the while. He was struck with the distinct manner in which he heard the beating of her heart and the convulsive sobs of her breathing—he listened to every sign as illustrating the principles of acoustics—and hence he assures us the origin of the mode of examining into diseases of the chest by auscultation now so generally adopted. M. Double evidently thought the anecdote redounded to his credit, but we fear he will look in vain for any compliment on the score of feeling—truly his sangfroid was indeed cold blooded."

Auenbrugger's description of percussion (1761) and especially Corvisart's translation (1809) of Auenbrugger's monograph popularizing the method were important in stimulating thought along lines of clinical method for detecting internal disease. It was almost certainly not a circumstance unrelated to the invention of the stethoscope that Corvisart was a teacher and colleague of Laennec. Observation had always been practiced by astute clinicians and palpation was becoming more extensively practiced. For example Morgagni (1114, vol. 1, p. 453) made reference to its use. Furthermore, in London, Matthew Baillie (1761–1823), nephew of the brothers Hunter and the man to whom John Forbes dedicated his translation of Laennec, taught and practiced palpation as part of the physical examination including that of the heart. In fact for the practice Baillie was criticized by Sir Henry Halford (1060) as treating his patients with unseemly familiarity! Laennec in the famous passage which will be quoted later makes reference to the fact that both palpation and percussion were used in the examination before invention of the stethoscope.

The fetal heart sounds were rediscovered by immediate auscultation before the stethoscope was invented or at least before publication of Laennec's monograph (1819). Laennec presented a preliminary report on the stethoscope to the Academy of Sciences in Paris in June 1818. On October 1, 1818, François Isaac Mayor (1779–1854), surgeon of Geneva, read a paper before the Society of Physical and Natural History of that city, and the following notice was recorded in the *Bibliotheque universelle*, which was a Readers Digest of the arts and sciences (1053).

"M. Mayor believes he has discovered something of importance to the art of obstetrics, namely, a means of determining whether the infant is dead or alive in the mother's womb; if the infant is alive, but the pelvis contracted, caesarean operation is performed. M. Mayor has demonstrated that when one places the ear to the abdomen of the woman the beating of the heart of the infant are heard." No formal communications were published. However, Mayor made other verbal reports in the following years. For example on July 8, 1819, before the same society he reported use of direct auscultation in the diagnosis of twin pregnancies (1152).

LAENNEC

René Théophile Hyacinthe Laennec (1781–1826) was a Breton who spent his professional career in Paris. Even without invention of the stethoscope he would deserve a place of honor in the annals of medicine for his pathological

studies, particularly those of cirrhosis, which bears his name, and of tuberculosis to which he himself succumbed at an early age As pointed out above, Laennec's was a prepared mind, as a result of the rediscovery of percussion by his teacher Corvisart and as a result of the practice of direct (immediate) auscultation by his good *friend and colleague Robert Bayle*

Laennec's account of the discovery of the method of stethoscopy is probably the most famous single passage in medical literature (831c) "I was consulted in 1816 by a girl who presented the general symptoms of heart disease and in whom palpation and percussion gave little information on account of the patient's obesity Her age and sex forebade an examination [by direct auscultation] Then I remembered a well *known acoustic fact, that if the ear be applied* to one end of a plank it is easy to hear a pin's scratching at the other end I conceived the possibility of employing this property of matter in the present case I took a quire of paper, rolled it very tight, and applied one end of the roll to the precordium, then inclining my ear to the other end, I was surprised and pleased to hear the beating of the heart much more clearly than if I had applied my ear directly to the chest "

Laennec's *Auscultation Médiate* first appeared in 1819 The three years following the first conception of stethoscopy in 1816 had been spent in intensive study of the method at the Necker Hospital in Paris and correlation of autopsy findings with clinical impressions A second edition appeared in 1826, within weeks of his death

Pulmonary disease *in general and respiratory* sound in particular clearly represented Laennec's "first love He made several major errors in interpreting cardiovascular sound He related the first sound ("bruit ventriculaire") to ventricular systole and the second sound ("bruit auriculaire") to atrial systole Because of this confusion he never was *able to relate murmurs* accurately to lesions of particular heart valves Laennec was not explicit as to how he thought contraction of the atrium and of the ventricle caused the heart sounds probably he thought the contracting myocardium developed vibrations within itself So often did autopsy reveal no cardiac lesion in patients in *whom he heard a* murmur, that he concluded *in his second edition* that spasm of the heart or great vessels ("une contraction spasmodique du coeur") was responsible for most or even all murmurs, thereby abandoning the view expressed in his first edition that valvular obstruction produced murmurs and thrills He never recognized the diagnostic significance of the pericardial friction rub even though Victor Collin, his *chef de clinique* (resident physician), gave a detailed description in 1824 in his doctorate thesis (280) ("There are few diseases more difficult to recognize than pericarditis I must acknowledge that mediate auscultation does not afford certain signs of pericarditis ") He heard what we call venous hum and compared it to the sound of the "sea or that produced by the application of a large sea shell to the ears" but he thought *the sound was arterial in origin Corrigan (296)* claimed that Laennec had no conception of the fact that murmurs and thrills are manifestations of one and the same vibratory phenomenon (see p 43)

Laennec introduced the terms "bruit de soufflet" (bellows murmur blowing murmur) and "bruit de lime bruit de rape" and "bruit de scie" (filing rasping, and sawing murmurs varieties of musical murmurs) It is likely that Laennec described the auscultatory sign of mediastinal emphysema which Hamman (see p 17) rediscovered over a century later He referred to the clicking sounds as "le râle crépitant sec à grosses bulles" (1370)

He wrote as follows "Sometimes, although very rarely, it happens, in connection with palpitations *that each contraction of the ventricle* is followed by several successive contractions of the auricles which together do not occupy more time than an ordinary single contraction Sometimes I have counted two pulsations of the auricles for one of the ventricles other times four but the most frequent number of the successive con*tractions corresponding to one contraction of the* ventricle was three I have observed them only in persons affected by ventricular hypertrophy Since Laennec considered the normal second sound to be related to contraction of the auricle he was probably dealing in this passage with a variety of states in which one or *more extra sounds occur at the end of systole*

or early in diastole (p 166) late systolic click, split second sound mitral opening snap early diastolic snap of constrictive pericarditis protodiastolic gallop Extrasystoles may have accounted for some of his case Clearly the contractions of the auricle cannot be taken literally as have some authors who *interpret this passage* as a description of atrial heart sounds with heart block

Laennec described noisy and musical cardiopulmonary murmurs In certain persons the anterior borders of the lung extend in front of the heart ... If one examines such a person when his heart is beating more forcibly than usual the diastole of the heart compressing these portions of the lungs and forcing the air out of them alters the breath sound in such a way that it imitates a blowing murmur or the sound of a wood file ... it disappears almost entirely when the patient is made to hold his breath for a few moments [1]

Laennec (831) first invented the term *pneumopericardium* and wrote as follows Sometimes the air is combined with a liquid and this is by much the most frequent case at other times the pericardium is distended by air alone ... I have sometimes been enabled to announce its presence from the supervention of an increased resonance over the lower part of the sternum and from the existence of the sound of fluctuation produced by the action of the heart and by deep inspirations ... I am convinced that in almost all the cases where the sound is heard at a distance the cause of the phenomenon is a temporary development of gas in the pericardium

The romance of Laennec's discovery and of his premature death from tuberculosis has captivated the imaginations of lay writers as well as of students of medicine and medical history In 1949 *Docteur Laennec* a reasonably documentary motion picture account of his life was a hit in Paris Kipling in a short story written in 1908 and entitled *Marklake Witches* has Laennec captured by the British in the Napoleonic Wars and billeted in a small English village (150) One day the heroine comes upon Jerry the local medico and Laennec (the two are the witches of the title) playing with toy trumpets They were not real trumpets because Jerry ... put his trumpet against René's collar and listened while René breathed and coughed Said Jerry 'Tis wonderfully like hearing a man's soul whispering in his inward but unless I'm a buzzing in my ears you make about the same kind o' noise as old Coffer Marklin—but not quite so loud as young Cooper It sounds like the breakers on a reef—a long way off Comprenny? Perfectly, answered *René He knew* the significance of these sounds in his chest, and in his soul he said *'I drive on the breakers* But before I strike I shall save hundreds thousands millions perhaps by my little trumpet'

¹ Quoted by Hirschfelder (630) p 114

THE STETHOSCOPE[1]

The well known acoustic fact' referred to by Laennec in the famous passage quoted earlier was known to Leonardo da Vinci (1452-1519) who described (1006) the forerunner of the stethoscope If you cause your ship to stop and place one end of an oar in the water and the other end to your ear you will hear ships at a great distance from you You can also do the same by placing the end on the ground and you will then hear anyone passing at a distance from you It is unlikely, however that da Vinci's observation was known to Laennec

Laennec described his own stethoscope the model which quickly replaced the rolled up 'quire of paper' in the following manner (831)

It consists simply of a cylinder of wood perforated in its centre longitudinally by a bore three lines in diameter and formed so as to come apart in the middle for the benefit of being more easily carried One extremity of the cylinder is hollowed out into the form of *a funnel to the* depth of an inch and half which cavity can be obliterated at *pleasure by* a piece of wood so constructed as to fit it exactly with the exception *of the central bore* which is continued through it so as to render the instrument in all cases a pervious tube The complete instrument —that is with the funnel shaped plug unfixed—is used

¹ (*Cf* Figures 4 and 5)

in exploring the signs obtained through the medium of the voice and the action of the heart, the other modification, or with the stopper removed, is for examining the sounds communicated by respiration" With his own hands Laennec lathed most of the first stethoscopes

Although mild objections and a certain amount of ridicule accompanied the early years of the



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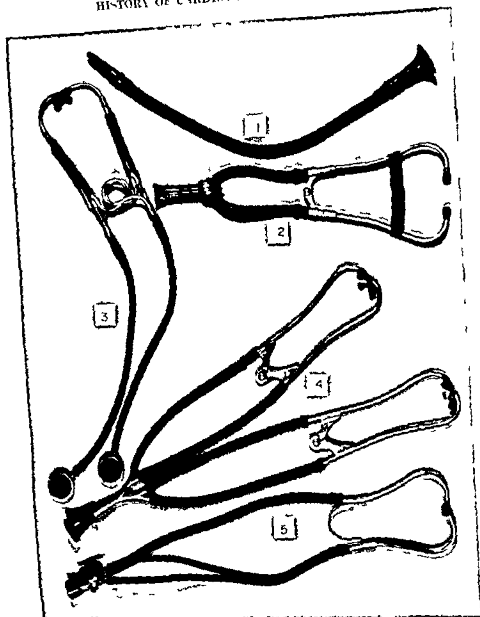


FIG 5 Flexible stethoscopes mostly binaural (Unless otherwise indicated exhibited through courtesy of Museum of Armed Forces Institute of Pathology) (1) Type used as early as 1830 chest piece at right end (2) Invented by George P Cammann New York (1855) (3) Kerr symballophone (Courtesy of Dr Robert W Caris Baltimore) (4) Teaching stethoscope double (before 1896) (5) Modern stethoscope with Sprague Bowles combination chest piece

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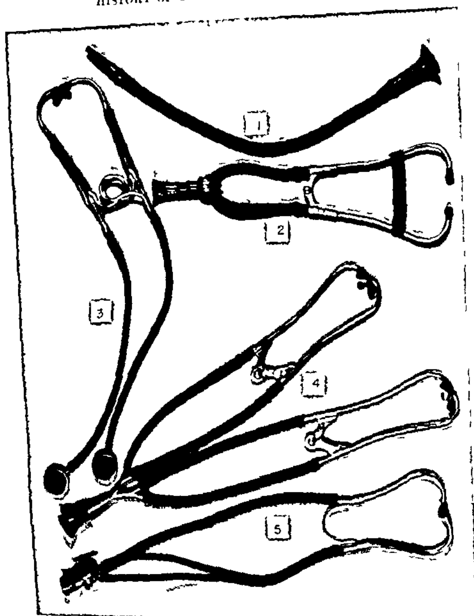


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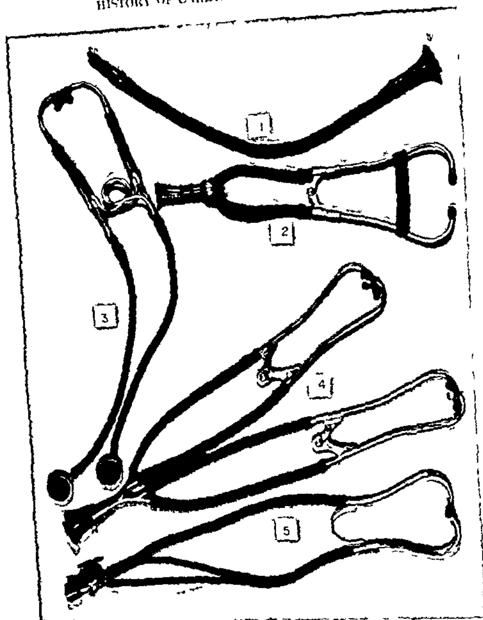


Fig. Flexible stethoscope mostly binaural (Unless otherwise indicated exhibited through courtesy of Museum of Armed Forces Institute of Pathology) (1) Type used as early as 1850 chest piece at right end (2) Invented by George P Cammann New York (1855) (3) Kerr symballophone (Courtesy of Dr Robert W Carns Baltimore) (4) Teaching stethoscope double (before 1890) (5) Modern stethoscope with Sprague Bowles combination chest-piece

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heart block and periodic breathing (also see p 22), published a small book entitled *On the Use of the Stethoscope*

Sir Charles Scudmore (1779–1849) of London returned from Paris with his initial reservations about the stethoscope changed into enthusiasm when Laennec, discovering that Scudmore's ear had an unusually large tragus, had the end of the stethoscope hollowed out to obtain a better seal (1565) In the clinic of Nasse (1778–1851), whose name is associated with the law describing the sex linked inheritance of hemophilia, the stethoscope was used early in Germany The large group of American students in Paris in the first half of the last century brought the gospel back with them W W Gerhard (1809–1872) of Philadelphia wrote a comprehensive text on percussion and auscultation Henry Ingersoll Bowditch (1808–1892) uncle of Henry Pickering Bowditch (1840–1911) the Harvard physiologist who discovered the "all or none" law of heart, wrote a manual called *The Young Stethoscopist* (1846), and Oliver Wendell Holmes (1809 1894) wrote both a prize winning Boylston essay (1836) and a satirical poem (p 52) on the new method

An interesting view of the reception of the stethoscope in London is provided by the London *Times* which on December 19 1824 carried the following piece (1310)

"A wonderful instrument called the Stethoscope invented a few months ago for the purpose of ascertaining the different stages of pulmonary affections is now in complete vogue in Paris It is merely a hollow wooden tube about a foot in length (a common flute with the holes stopped and the top open would do perhaps just as well) One end is applied to the breast of the patient the other to the ear of the physician, and according to the different sounds harsh, hollow soft loud etc he judges of the state of the disease It is quite a fashion if a person complains of a cough to have recourse to the miraculous tube, which, however cannot effect a cure, but should you unfortunately perceive in the countenance of the Doctor, that he fancies certain symptoms exist it is very likely that a nervous person might become seriously indisposed and convert the supposition into reality "

It is not improbable that the comment of the London *Times* was influenced by John Forbes' mistaken, indeed silly, evaluation in the translator's preface to the 1821 English edition of Laennec (468)

That it will ever come into general use notwithstanding its value I am extremely doubtful because its beneficial application requires much time and gives a good bit of trouble both to the patient and the practitioner, because its whole hue and character are foreign and opposed to all our habits and associations It must be confessed that there is something even ludicrous in the picture of a grave physician proudly listening through a long tube applied to the patient's thorax as if the disease were a living being that could communicate its condition to sense within

Pierre Adolphe Piorry (1794–1879) who advocated the use of a plessimeter (or pleximeter) in percussion so called mediate percussion "slenderized" the stethoscope by reducing its diameter to that of a finger (1211) He also introduced the trumpet chest piece and improved the ear piece so that better seal was attained (1243) He introduced a removable ear piece for greater portability His was the stethoscope used in France—when any was used—for the next 75 to 80 years

C J B Williams (1805–1889) is said by his son (1565) to have determined that a flexible stethoscope was as satisfactory as the solid type In fact in the 1830's Williams stated the following in an article for the *Cyclopedia of Practical Medicine*

The flexible caoutchouc tube terminating in a small ivory funnel now in common use by deaf persons is employed by some auscultators and in some respects answers the purpose well enough but it is in others decidedly inferior to the solid instrument

In 1839 Charcellay of Tours (250) in passing pointed out the advantage of the flexible stethoscope in permitting simultaneous observation of the pulsations in the neck and auscultation of the precordium It was in the analysis of what we would now call diastolic gallops that Charcellay found it useful

In *The Young Stethoscopist* (1846) Bowditch refers to the monaural *flexible stethoscope used* by Caspar Wistar Pennock of Philadelphia In 1844 Charles Goodyear developed the method

for improving rubber by addition of sulfur to the native product. This technologic development paved the way for the flexible stethoscope.

American physicians can take pride in the fact that the binaural flexible stethoscope originated in this country. George P. Cammann (1804–1863) is usually credited with its invention. By 1852 he had already developed a stethoscope with flexible tubing (spirals of wire covered with silk, later rubber, or as he called it, caoutchouc), ivory ear pieces, and a spring cross piece to hold the ear pieces in place. The formal announcement of this stethoscope appeared in 1855 (223). In 1861 S. S. Alison in his *Physical Examination of the Chest* (p. 324) pictured the Cammann stethoscope which is almost precisely like the modern binaural stethoscope with bell chest piece.

Curiously enough, in France, Laennec's homeland, the stethoscope was slow in gaining wide use and direct auscultation was generally practiced instead of stethoscopy. Rist, who was prominent in introducing the binaural stethoscope into France, is quoted (187) as making the following statement about 1935: "Until twenty five years ago immediate auscultation was used almost exclusively in France. The binaural stethoscope was absolutely unknown. The monaural was on the table of every practitioner but seldom used, being so utterly impractical. I was converted to the use of the binaural stethoscope on my first journey to the United States in 1908, and have demonstrated it to my students since that time, while my colleagues looked at me with awe and thought I was making myself ridiculous." In 1936 Professor Étienne Bernard (91) wrote that "up until a relatively recent date most practitioners in France auscultated patients by applying the ear directly to a thin handkerchief placed on the chest of the patient. However, since a time which can be fixed at about 20 to 25 years ago, use of the binaural stethoscope has become more and more widespread and it can be said that at present most practitioners use it." It was the advent of the auscultatory method for measuring blood pressure which made the binaural stethoscope unavoidable.

The important studies of the giants of French cardiology such as Duroziez (see p. 27) and Potain (see p. 21) were done by direct auscultation or by means of the monaural stethoscope. In 1957 Charles Laubry (see p. 26) wrote me that he never saw his old chief Vaquez use a binaural stethoscope until about 1915 or 1916.

The combination of the prevailing practice of direct auscultation by the French professors with the Frenchman's particular brand of humor is doubtless responsible for numerous cartoons satirizing physicians (see Fig. 26).

There have always been those who champion the superiority of the naked ear, at least for some varieties of sound. Lewis A. Conner (1866–1950) of New York City, a founder of the American Heart Association and one of the first editors of the American Heart Journal, pointed out that faint, high pitched aortic diastolic murmurs are sometimes better heard by direct auscultation than by stethoscopy (282). He is said always to have carried a silk handkerchief for use in direct auscultation.

There have also been those who preferred the monaural solid stethoscope to the flexible binaural instrument. Writing in *Lancet* in 1902 (1457) one Syers stated: "I look upon the use of the binaural stethoscope as being in every way most objectionable. . . . The double stethoscope should be altogether done away with and abolished from the face of the earth—Delenda est Carthago." To this very day there are many parts of the world, including most of central Europe, where the monaural stethoscope is used. It is the teaching in the medical schools in these parts that one "hears too much" with the binaural stethoscope and is likely to be confused thereby. Pediatricians are usually the only ones to use the binaural flexible stethoscope, the advantages being that it is possible to follow the movements of the child and extraneous noise is better excluded. It is interesting to find the same evaluation of the relative merits of the two types of stethoscope in Sansom's *Physical Diagnosis of the Heart*, 1881 (p. 92).

The next main development in the direct phylogeny of the modern stethoscope was the diaphragm (or membrane) chest piece, usually attributed (187) to R. C. M. Bowles, an engineer of Brookline, Massachusetts, who patented the

diaphragm in 1894. When it was recognized that both the bell and the diaphragm have merit and in effect complement each other, a composite chest piece permitting rapid change from one to the other by means of a valve was the next advance. In 1926 Howard B. Sprague (1895–) of the Massachusetts General Hospital, Boston, introduced the combination chest piece (1428) now in general use.

Studies of stethoscope function have been conducted by several workers, particularly Rappaport and Sprague (1244, 1245), and have defined proper length and bore of tubing (1243), the importance of snug fit of the ear pieces (1242), the distinctive function of the bell and diaphragmatic chest pieces (1245), the proper structure for these chest pieces.

Several models and variations of the stethoscope have not survived. In 1859 Alison (15) invented a stethoscope in which the chest piece was a small sac of water. He suggested that improved coupling with the chest, including close fit between ribs, might improve audibility of chest sounds. The phonendoscope (99) invented by Bazzi, a physicist, and Bianchi, a physician, of Parma, Italy, in the 1890's is another example of an obsolete stethoscope (Fig. 6, A and B). A small knob on the end of a rod was applied to the chest. The other end of the knob was attached to the center of a diaphragm. A second diaphragm was mounted parallel to the first with only a small air space between the two. The second diaphragm was comparable to the ordinary single diaphragm chest piece. The advantages of the phonendoscope were said to be precise localization of sounds and amplification of faint sounds. The idea of a stiff diaphragm is, perhaps, the only surviving feature of the phonendoscope. Parker's refractoscope (1181) made use of a resonating cavity whose volume could be varied.

In 1884, one Aydon Smith (1405) quite seriously recommended an instrument which could be used as a monaural, binaural, or differential stethoscope; the tubing could serve as a tourniquet, catheter or stomach tube, and the chest pieces as an otoscopic speculum or funnel for administering fluids through a tube! Enemas or douches could also be administered.

Differential stethoscopes of various types were designed. In 1861 Alison discussed those in existence at that time (16). The principal differential stethoscope designed in recent years is that of Dr. William Kerr (1889–) of San Francisco, formerly Professor of Medicine at the University of California (785). The Kerr symballophone (Fig. 5) is a double stethoscope as far as chest pieces are concerned. Sounds picked up by each chest piece are led into both ears. The advantage is said to be ease of comparing the sounds in two areas of the precordium or of the lung fields.

The electric stethoscope for multiple student teaching and for aid to the deaf physician was a logical development paralleling those in the field of hearing aids, telephony and electronics. Cabot's, dating from the early 1920's, is one of the first examples (206).

The stethoscope has, of course, become virtually



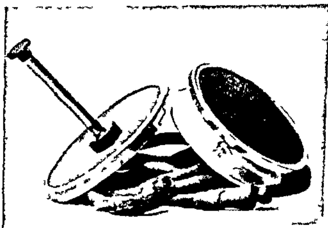


Fig. 6. The phonendoscope of Bazzi and Bianchi. (From Bianchi's *monograph* (99).)

the symbol of the medical profession Dr Arthur Conan Doyle (1859–1930) has his Sherlock Holmes make indirect use of the stethoscope in identification

As to your practice if a gentleman walks into my rooms smelling of iodoform with a black mark of nitrate of silver upon his right forefinger and a *bulge on the side* of his top-hat to show where he has secreted his stethoscope I must be dull indeed if I do not pronounce him to be an active member of the medical profession

—*A Scandal in Bohemia* Chapter 1

THE GOLDEN CENTURY OF STETHOSCOPY (1819–1919)

There is nothing so captivating as new knowledge Even though its subject be incurable diseases which it renders not a whit the less incurable still it is captivating Cases of such diseases [i e with murmurs] always abounded in hospital They were essentially difficult cases Their symptoms were hard to interpret into any definite meaning They betokened that in some way or other the heart was diseased and that in some way or other their termination was death Day by day to watch over these cases and to treat them was an irksome duty—it was even a thing to damp the spirit

But auscultation brought to them a new light and a *new interest* And then these same became the cases which we were continually busy about which we were never tired of visiting and examining and auscultating and of examining and auscultating again and again

—Latham *Lectures* 1847 (343 p 53)

The golden century of *stethoscopy* began with Laennec and can be said to have ended with Sir James Mackenzie It was the age of Bouillaud Hope Flint Duroziez and Potain to mention only five among many It was a period of exciting exploration and occasionally extravagant claim Virtually *all the* phenomenology of cardiovascular sound was described in this period It can be considered to have ended in 1908 with the first edition of Mackenzie's *Diseases of the Heart* It ended then in part because Mackenzie de-emphasized valvular disease *per se* and decried *the grave* prognostic significance that was then *usually* attached to any murmur in part because about 1908 the age of stethoscopy was *replaced* by the *Age of Phonocardiography* Since 1908 virtually all studies of cardiovascular sound have had phonocardiographic documentation although a few strictly stethoscopic studies of importance have continued to appear

The several workers in the golden century of stethoscopy and their contributions will be considered in alphabetical order Some liberty with time has been taken in as much as two or three persons whose contributions were made later than a century after Laennec are included

Jean Baptiste Bouillaud (1797–1881) Paris (Fig 7) wrote extensively on cardiovascular sound in his *Traité Clinique des Maladies du Coeur* of which the first edition appeared in 1835 He is said (1224 p 34 *ff*) to have described and named *bruit de galop* (gallop rhythm) in 1847 He assigned the designation *bruit de rappel* (a particular type of drum beat) to a group of conditions including split second sound and mitral opening snap He also compared the sound (140 p 213) to that of the hammer *which after striking the iron falls on the anvil* rebound and fall again *motionless* Bouillaud described venous hum under the designation *bruit de diable*[4] diable being the name of a French toy like a humming top He noted its frequent occurrence with anemia[5] He described the musical type of venous hum and used musical notation to represent examples in his textbook (140 p 264) However like Laennec he assigned its origin to the carotid arteries interestingly he called the musical venous hum *chant des artères*

The following passage is selected from among Bouillaud's many observations on cardiovascular sound (140 p 387) I have encountered a large number of cases where the valvular sounds had a timbre so dry, so snapping and so hard that one would think he was hearing the sound which two sheets of parchment produce in striking each other abruptly and forcefully hence the name snap (*claquement*) or parchment sound (*bruit de parchemin*) by which I have *been in* the habit of designating this modification of the valvular *sound* Bouillaud related his parchment sound to fibrosis in the valve Bouillaud also described a quadruple movement and quadruple heart sound *rhythme à quatre*

[4] Writing in 1884 Alonzo Clark (1807–1887) humorously stated that a friend of his translated this freely as a devil of a noise (1767)

[5] The Germans called it *Nonnensausen* nun's murmur possibly because it is more frequent in the *menstruating* and *notorious* it undernourished females



Fig 7 *(Upper left)* Bouillaud *(upper right)* Fauvel *(lower left)* Hope *(lower right)* Hope's own drawing of his case of aneurysm of the aortic sinus of Valsalva ruptured into the right ventricle From 1839 (3rd) edition of his *Diseases of the Heart*

temps in which the two intermediate sounds follow each other in quick succession and resemble a small dry crack." The basis was, he suggested "a large, broad, calcified plaque forming an embossment on the surface of the heart" found at autopsy

Richard C Cabot (1868–1939) Boston described (with Locke) in three cases of intense anemia a diastolic murmur which was loudest at the fourth left costal cartilage and suggested aortic regurgitation *(209)* *But at autopsy in all* four the aortic valve was sound Later Cabot

and colleagues made pioneer studies of the frequency composition of heart and lung sounds (208). The first edition of Cabot's *Physical Diagnosis* appeared in 1900.

In 1832 *Dominic John Corrigan* (1802–1880) of Dublin wrote on "permanent patency of the mouth of the aorta or inadequacy of the aortic valve" (295). The characteristic quality of the pulse since called by his name was pointed out and the murmur of aortic regurgitation was partially described. His pioneer studies on the mechanism of murmurs and thrills (296) are described on page 43.

John Elliotson (1791–1868) of London, first professor of medicine, University College (1823), was an imaginative person, a minor genius, an astute observer. He called attention to Robert Hooke's reference to the heart sounds and prediction of stethoscopy. In the Lumleian lectures of 1830 he described four cases of musical murmurs: "I have heard it [the musical murmur] exactly resembling the cooing of a dove—a variety not mentioned, I believe, by authors." He discovered and fostered James Hope. Later Elliotson lost practice and prestige because of his work with hypnosis and the treatment of hysteria. He was one of the first to do surgical procedures under hypnosis and he was undoubtedly ahead of his time, but the methods he used for publicity turned the profession against him.

John D. Fisher (1797–1850) of Boston described cephalic murmurs (460).

Louis Gallavardin (1875–) Lyons cardiologist (487) wrote about systolic clicks, referring to the phenomenon as *bruit de triolet* and presented evidence for their origin in pleuropericardial adhesions (514). In calcific aortic stenosis he (521) described the dissociation between a noisy murmur in the aortic area and a musical murmur at the left sternal border and apex. He (514, 515) emphasized the simulation of mitral stenosis where a late systolic click occurs. He (511) called the sounds audible with atrial systole in heart block *galop de bloc*, believing them due not to atrial contraction itself but rather to repercussion in the ventricle of the atrial contraction. A son, Leon Gallavardin, working particularly in collaboration with Roger Froment (488–491, 494, 495) has made contributions to phonocardiography.

Robert James Graves (1797–1853) of Dublin helped establish the friction rub as a sign of acute pericarditis (586). He also described musical pericardial friction sounds which he said resembled the sound of a wet finger rubbing on glass. He probably described the benign though loud and weird-sounding musical murmur of pericardial origin heard sometimes, especially in young persons with febrile illness, and sometimes persisting for months or years (patient Mary Robin on 586 p 926).

Graves, in describing the condition which bears his name, stated that at times the accentuation of the heart sounds in thyrotoxicosis is so marked as to render the first sound audible at a distance from the chest:

> I have lately seen three cases of violent and long continued palpitations in females, in each of which the same peculiarity presented itself, viz. enlargement of the thyroid gland; the size of this gland at all times considerably greater than natural, was subject to remarkable variations in every one of these patients. The palpitations have in all lasted considerably more than a year, and with such violence as to be at times exceedingly distressing, and yet there seems no certain grounds for concluding that organic disease of the heart exists. In one the beating of the heart could be heard during the paroxysm at some distance from the bed, a phenomenon I had never before witnessed, and which strongly excited my attention and curiosity. She herself, her friends, and Dr. Harvey all testified the frequency of this occurrence and said that the sound was at times much louder than when I examined the patient, and yet I could distinctly hear the heart beating when my ear was distant at least four feet from her chest! It was the first or dull sound which was thus audible.

This observation recalls Bartholin's observation (p 5). It also brings to mind a veterinary experience. A cow developed angioneurotic edema with laryngeal edema and probably asthma. After intravenous injection of what seemed an enormous dose of an adrenaline-like material the respiratory distress was promptly relieved but the heart sounds were clearly audible at a distance of several feet for about fifteen minutes.

Louis Virgil Hamman (1877–1946) of Baltimore described the aortic diastolic murmur which may accompany dissecting aneurysm of the aorta and attributed it to distortion of the aortic

ring by the hematoma of the media (628) This sign had been described by Letulle in France in 1905 (884) and first in the English language literature in 1926 by William H Resnik and Chester S Keefer (1262), then assistant resident physicians at the Johns Hopkins Hospital The latter writers thought, however, that the phenomenon was due to backflow of blood through the false channel In the introduction to the clinical pathological conference in which Hamman emphasized the sign of aortic regurgitation in dissecting aneurysm, he gave his charmingly expressed appraisal of the "C P C " as a teaching exercise (628)

> it seems to me a delightful and entertaining exercise and one not altogether without profit As a method of instruction it has great advantages over the bedside clinic not, it is sure, to the physician discussing the problem, but certainly to his hearers At the bedside an able and experienced physician supported by the weight of reputation may make almost what he pleases of the clinical facts before him None will be so rash as openly to dispute his conclusions and nearly all will go away convinced of his skill and erudition But his position at a clinical pathological conference is quite different Here all the advantage is on the side of the hearer for though the physician may be supported by a reputation to equal Osler's and by the honors of all the Academies there sits the smiling pathologist ready and sometimes even eager to administer the *coup de grace* to his reputation erudition and eloquence What he may say is to be judged immediately and irrevocably He is no longer the glorious high priest of medical science introducing novices to her mysteries but the very humblest suppliant prostrate at her feet

Hamman also wrote about spontaneous mediastinal emphysema (Hamman's disease) and the adventitious sounds which are associated with it (630) (631) (632)

In 1828 *Thomas Hodgkin* (1798–1866) of Guy's Hospital London, and Hodgkin's disease fame described (694) a musical murmur in association with what he called retroversion of an aortic cusp and further defined as a particular state of the valves of the aorta, which by admitting of their falling back towards the ventricle unfits them for the performance of their function He called the murmur *bruit de scie*— a purring thrilling, or sawing kind of noise " Hodgkin himself attributed the original observation to Key (1827) and the phenomenon in question is sometimes called the Hodgkin Key murmur

In his short lifetime (738A) *James Hope* (1801–1841) made many important contributions to the clinical phenomenology of cardiovascular sound and did animal experiments as well (Fig 7) His personality was marred by contentiousness of pathologic proportions and a concern about priorities which amounted to paranoia In particular, C J B Williams (p 23) and Bouillaud seem to have been objects of Hope's antipathy Hope's textbook *Diseases of the Heart* is perhaps the first English text of cardiology in the modern sense The first edition appeared in 1832, the third edition (1839) is particularly noteworthy and is both interesting and instructive to the modern reader, although as Latham commented in 1847 " its style, which is too often controversial, and even disputatious repels many readers, and has been in some measure a hindrance to its usefulness (711, p 470) For example, in the preface of the 1839 edition he wrote 'I have ventured to reclaim for my countrymen and self a number of discoveries which an eminent French writer [probably Bouillaud], probably from unacquaintance with the English language and medical literature, has imagined to have emanated from himself " When he performed experiments he recorded with utmost care the precise date and the name of the individuals who observed them he would even provide each observer with a protocol of the experiments to be performed mainly so that there could be no question about whose were the ideas behind the experiment

Hope invented the term venous hum ' and supported the contention of Ogier Ward of Birmingham that the sound arises in the jugular veins not the carotid arteries as held by Laennec and Bouillaud By the management of pressure with the stethoscope over or near large veins the venous murmur may often be raised by a gradual swelling into a more or less musical hum such as is yielded by a child's humming top I propose to denominate this *Venous Hum* for without being unnecessarily queamish I think that this is not only a rather more euphonious epithet but more intelligible than noise of the devil by which term derived from a

plaything [spinning top] known to few. M. Bouillaud has designated the hum in question (711 p 118)

Hope described the continuous murmur of aorto-pulmonary communication in a case of rupture of an aortic aneurysm into the pulmonary artery. It appears that a *continuous* murmur extended from the first over the second sound (711 p 470) He found a similar murmur with rupture of a sinus of Valsalva into the right ventricle. The last condition was illustrated with a drawing by Hope himself (Fig 7)

Hope described the cardiopulmonary murmur in two students of University College. Both wore very tight waistcoats preventing the expansion of the lower ribs. During this state of breathing ... a slight bellows murmur with the first sound over the semilunar valves existed in both. It was not however exactly synchronous with that sound but began an instant later as if from a separate cause. In both the murmur ceased entirely when unbuttoning their waistcoats and waistbands of their trowsers they breathed with the lungs naturally inflated. By alternating the circumstance the murmur could be created or removed at pleasure. I presume therefore that it proceeded from a cause exterior to the heart (711 p 391) Movement of air in compressed lung was suggested as the mechanism of the murmur

In discussing musical murmurs Hope wrote "The musical tone was a clear note like the oo of *coo*... *welling* and also rising a semitone in the *middle like the mew* of a kitten. It attended the second sound and proceeded from aortic regurgitation (711 p 87) Hope noted the role of a vibrant member in the production of musical murmur — presentation of an edge to a stream is best calculated ... to produce musical vibration ... He also made the analogy *to various* musical tones — there is but a shade of difference in the mechanism by which we make the *lips* produce a blow or a whistle the latter *depending* on the happy and steady *adaptation of the size* of the aperture to the *strength of the* current

Hope produced hemic murmurs in dogs by copious bleeding. What was known as Hope's early diastolic murmur was probably the Graham Steell murmur (see p 30)

An anonymous poet inscribed the following verse in a copy of Hope's textbook in the library of the Royal College of Physicians London (1164)

He opened wide the portals of the heart
And bade us hearken to its varied beat
Taught us the rules of stethoscopic art
And showed disease slow running in its seat

Henri Huchard (1844–1910) of Paris described the musical murmur which may *be associated* with aberrant tendon of the left ventricle (715 718) atrial heart sounds (717) with complete heart block (*systoles en echo*) and variation of the first heart sound in complete heart block (717) with occasional very loud sound (*bruit de canon*). The clinical picture of aberrant tendon of the ventricle was described on the basis of a 49 year old patient with nephritis and cardiac hypertrophy. As well as a musical systolic murmur there was a presystolic gallop. At autopsy there was ... other than left ventricular dilation and hypertrophy no lesion except an aberrant tendon (see Fig 184). Presumably the aberrant tendon a congenital malformation gave no clue to its presence until dilatation of the ventricle pulled it taut so that it vibrated musically during ventricular ejection

In one patient a 70 year old hypertensive with no peripheral signs of aortic regurgitation Huchard (716) described an *exceedingly loud* musical diastolic murmur which immediately suggested to observers including Duroziez rupture valvulaire des sigmoide ... No valvular lesion was discovered at *necropsy*. *Huchard him*self thought the *murmur probably cardiopulmonary*. Pericardial *origin* is also possible. Least likely is a very *slight but noisy leak* at the aortic valve

In his lectures on heart disease (1847) *Peter Mere Latham* (1789–1875) of St Bartholomew's Hospital London a magnificent clinical teacher organized well the practical aspect of auscultation. His superb lectures were rendered *memorable* by the frequent use of epigram bon mots *aphorisms and* well expressed common sense

Some of his contemporaries less competent and less discreet may have overdone the embellishments in lecturing and ward rounding and

even Latham's approach may have been unattractive to some. In 1833 Henry Ingersoll Bowditch (see p. 12), then aged 25 years and fresh from the service of Louis and others in Paris, wrote as follows about his visit in London: "I followed Dr ——————, one of the chief physicians of London, in his visit at the hospital. . . . His main object seemed to be to make the students laugh. I was completely disgusted. Such is the case with most of them. They talk much but know little" (144).

In discussing factors determining the intensity of murmurs, Latham pointed out (813, p. 37) the usually great intensity of musical murmurs, remarking on "a peculiar quality of the endocardial murmur, giving it a high musical note. Such a murmur will sometimes refuse to suffer restriction to a certain space within the body. It will even carry itself outwards and reach the ears of bystanders at a short distance." Latham was instrumental in the introduction of English words such as *murmur* in place of the French equivalents such as *bruit de soufflet*.

Jean Alexander Lejumeau de Kergaradec (1787–1877), Paris, made stethoscopic studies of fetal heart sounds, uterine souffle, and other acoustic phenomena of pregnancy. The findings were published in 1822 in Magendie's *Journal de physiologie expérimentale et pathologique* (872). Because of this excellent report Kergaradec rather than Mayor (see p. 7) long received credit for discovery of the fetal heart sounds. It is in fact likely that Mayor's observations were unknown to Kergaradec and certainly Kergaradec was the person who placed the acoustic phenomena of pregnancy at the disposal of the profession (24).

Samuel A. Levine (1891–) of the Peter Bent Brigham Hospital, Boston, has been a leading teacher of cardiac auscultation. In 1933 with Freeman (481) he suggested a system for the clinical grading of the intensity of murmurs. He has emphasized the relation between PR interval and intensity of the first heart sound and the usefulness of auscultation in the diagnosis of arrhythmias. He collaborated with W. P. Harvey in *Clinical Auscultation of the Heart* (1949).

Sir James Mackenzie (1853–1925) of Burnley, London and St. Andrews (Fig. 10), pointed out that the presystolic murmur of mitral stenosis disappears when the cardiac rhythm becomes totally irregular (1011). He stated that an explanation for this first occurred to him in 1897. He referred to auricular fibrillation as nodal rhythm because he found from recordings of the venous pulse and other beats that there was no evidence of atrial activity in such cases—he assumed that the pacemaker had moved to the atrioventricular node. At any rate, he assigned the correct explanation to the disappearance of the presystolic murmur, namely, that atrial systole was no longer occurring and that the atrium was in effect paralyzed.

Mackenzie emphasized the importance of the state of the myocardium as opposed to valvular disease. He decried the grim prognoses which at that time were often rendered on the basis of murmurs alone. In particular he emphasized the benignity of the apical systolic murmur when it occurred as an isolated finding.

The main contributions of *Thomas Bevill Peacock* (1812–1882) of St. Thomas' Hospital, London (Fig. 8), were descriptions of the findings of necropsy which corresponded to auscultatory findings in life. He wrote two monographs—*Valvular Disease of the Heart* (1865) and *Malformations of the Human Heart* (1858 and 1866)—and contributed many articles to the Transactions of the Pathological Society of London.

In 1854 Peacock described a musical diastolic murmur in a 64-year-old patient (1194). "It exactly resembled the sound produced by the common cuckoo clock . . . and was so loud as to be heard at a distance of several feet." The aortic cusps were thickened and separated with retroversion of the free edge of the right cusp. Although the valve lesion may have been syphilitic, the double systolic-diastolic character of the musical murmur and the relatively advanced age of the patient suggests atherosclerotic disease of the valve. (See p. 232 for discussion and Fig. 273 for illustration of a probably similar "cuckoo" murmur of calcific aortic valve disease.) In 1863 Peacock (1196) reported on a large number of patients with dissecting aneurysm of the aorta. Although he was one of the first to recognize that survival for years occurred in some patients



Fig 8 (*Upper left*) Potain (from biography by T   ter) (*upper right*) Duroziez (*lower left*) Thayer (*lower right*) Peacock

he did not note aortic regurgitation in any (p 452) Peacock (119, p 113) recognized the murmur of congenital pulmonary stenosis

Throughout his career *Pierre Carl Edouard Potain* (1825-1901) of the Charité Paris (Fig 8) devoted his major attention to the clinical study of cardiovascular sound His doctorate thesis in 1853 was devoted to the subject of the abnormal vascular bruit which follow hemorrhages (1227) His studies of gallop published in 1875 were outstanding (1225) He studied the presystolic gallop in particular referring to it as false reduplication of the first sound and indicated its association with contracted kidney —the basis for the designation *bruit de brightique*[a] He described the gallop in this manner This sound is much duller than the normal sound it is a shock a distinct pulsation scarcely a sound When the ear is applied to the chest it affects the sense of touch more perhaps than the sense of hearing and on using a flexible stethoscope it nearly always disappears altogether Potain held to the view that gallops are thud resulting from sudden filling of the ventricle

[a] *Nierengalopp* (kidney gallop) was the German term

He expounded the important concept that gallop sounds are an exaggeration of normal elements. 'If one auscultates a goodly number of healthy persons, one will not be long in discovering that diastole is not always absolutely silent; that in the part of the cardiac cycle where the abnormal sound responsible for the gallop is located, there is sometimes already, in vestigial form, something which, where exaggerated, could become the sound in question.' Dock (357, p. 36) wrote as follows:

> Potain and his pupils . . . made the French and Spanish speaking doctors acutely aware of the sounds in diastole which occurred with failure of the myocardium, but in the English speaking world these sounds . . . were either ignored or passed over lightly. Murmurs were the abnormality sought. . . . The *Osler* of 1920 has 'galloping consumption' but not 'gallop rhythm' in its index.

Although Schafer is said to have pointed it out in 1858 (1355), Potain (1226) in 1866 clearly described normal inspiratory splitting of the second heart sound. He described the early systolic click and attributed it to disease of the pulmonary artery or aorta; the midsystolic click and attributed it to pleuropericardial adhesions. Potain assigned the term *bruit de tabourka* to the ringing aortic second sound in conditions such as luetic aortitis.

In 1856 (1223) Potain described the early diastolic snapping sound of constrictive pericarditis.[7] 'The second sound was reduplicated or rather composed of two sounds following one another in quick succession: one clear, dry, ringing, with the usual characteristics of a valvular snap and of more or less unchanging intensity; the other coming immediately afterwards, less well defined, less resonant, coinciding exactly with the impulse felt in the precordial region, like the impulse increasing during inspiration, and taking on at that point a rather metallic tone.' The "impulse" is the *Spitzenstoss* of Skoda (1396), translated as "diastolic heart beat" by Francis C. Wood of Philadelphia (1587), a characteristic of constrictive pericarditis.

Potain (1224) maintained that all circumscribed systolic murmurs, be they early, mid, or late systolic, are non organic as compared to the murmur of mitral regurgitation which is holosystolic.

[7] Translation by Mounsey (1122).

Potain is also credited with invention of the dilution pipette for blood cell counts and of a sphygmomanometer with which he made pioneer observations on hypertension. About 1867 he attempted to record the heart sounds by means of tambours but discovered, of course, that the vibrations are of too low amplitude.

Josef Skoda (1805–1881) (actually Škoda, pronounced Schkoda), Professor of Medicine in Vienna, was a close clinical collaborator of the pathologist Rokitansky (a Czech like himself) at the Allgemeines Krankenhaus. Skoda was born in Pilsen, Czechoslovakia, where his brother Johann founded the well known Skoda works. The first edition of Skoda's textbook on percussion and auscultation appeared in 1839. Skoda emphasized the frequent occurrence of faint aortic heart sounds and accentuated pulmonary second sound in mitral stenosis—sometimes in the past called Skoda's sign (1395).

George F. Still (1868–1941), pediatrician of the Great Ormond St. Hospital, London, for whom Still's disease is named, described a musical variety of functional murmur in children often known as the "twanging string" murmur (1446):

> And here I should like to draw attention to a particular bruit which has somewhat of a musical character, but is neither of sinister omen nor does it indicate endocarditis of any sort. In my own notebooks I am in the habit of labelling it 'physiological bruit,' but only for want of some better name. It is heard usually just below the level of the nipple and about half way between the left margin of the sternum and the vertical nipple line. . . . It is systolic and often so small that only a careful observer would detect it; moreover, it is sometimes very variable in audibility, being scarcely noticeable with some beats and easily heard with others. Its characteristic feature is a twanging sound very like that made by twanging a piece of tense string. This bruit is found mostly between the ages of two and six years; as a rule they are brought for some ailment, such as a cough or some indigestion, and the bruit is discovered only in the course of routine examination. . . . It persists sometimes for many months. I have noted it as present in one case for two years. Whatever may be its origin, I think it is clearly functional, that is to say, not due to any organic disease either congenital or acquired.

William Stokes (1804–1878) of Dublin (see p. 11) made many clinical observations on cardio

vascular sound In his textbook *Diseases of the Heart and Aorta* (1855) Stokes commented on two cases with "extensive and irregular ossification of the aortic orifice" and murmurs audible at a distance—"at least three feet" in the case of one He noted that the very loud sound might be accompanied by little cardiac embarrassment

The perception of these sounds was the principal cause of his suffering for his general health long continued excellent and the heart's action was but little excited This gentleman once observed to me that his entire body was one humming top

The humming was audible over the extremities in this case which was apparently one of calcific aortic stenosis

William Sidney Thayer (1864–1932) of Baltimore (Fig 8) made early studies of the physiologic third heart sound (1464) which with Hirschfelder (690) he discovered about the same time as Gibson of Edinburgh and about two years after Obrastzow of Kiev He was impressed with the accentuated third sound of mitral regurgitation (1465) He pointed out that the third heart sound was loudest immediately after the subject assumed the recumbent posture—so called primo-decubitus position (1468) Thayer thought the third sound was due to the sudden tensing of the mitral and perhaps at times the tricuspid valves at the time of the first and most rapid phase of diastole (1464) He introduced the term opening snap to correspond to the claquement d'ouverture of Rouches (1467) He was early (1911) to describe an epigastric venous hum with hepatic cirrhosis (1466) With the pathologist William George MacCallum Thayer made studies of experimental aortic regurgitation in dogs (1471)

Walter Hayle Walshe (1812–1891) after C J B Williams Professor of Medicine at University College Hospital made many observations on cardiovascular sound He noted the change in quality of aortic murmurs on transmission to the apex (1505)

Charles J B Williams (1805–1889) succeeded Elliotson as Professor of Medicine at University College William invented the onomatopoetic lubb tup (1563) He appears to have described atrial sound in a case of complete heart block (1563) In studies sponsored by the British Association for the Advancement of Science he devised the method of relating valve action to heart sound by putting his finger into the atrium of animals via the auricular appendage (1562) Of recent years this technique has been commonly used in mitral valve surgery

MITRAL STENOSIS (67 462 1509) Writing in 1950 Cabo (221) made the following observation

Because of the multiplicity of its auscultatory signs because of the unusual character of certain of these and finally because of its own frequency mitral stenosis probably is the cardiopathy which has motivated the largest number of clinical and phonocardiographic studies For these reasons the history of this condition will be separately traced

As we have seen earlier (p 6) *Corvisart* may have described the diastolic murmur and thrill of mitral stenosis Laennec was probably familiar with both to some extent In his 1826 edition he described the case of Louis Ponsard a 16 year-old gardener whom he had seen in 1822 The contraction of the auricle was exceedingly prolonged took place with a dull bruit very strong and quite like the sound produced by a file rubbing on wood This bruit was accompanied by a purring sound which was heard by the ear and was evidently the same as felt by the hand[a] At the end of the contraction one heard a loud bruit accompanying the impulse and synchronous with the pulse Since he thought the second sound represented the contraction of the atrium the prolongation of which he speaks was undoubtedly the diastolic rumble of mitral stenosis ending in a loud mitral first sound

In 1824 *J R Bertin* (1767–1828) of Paris wrote as follows in his text on cardiac disease (95)

49th observation Bellows murmur during the contraction of the auricle Fibro cartilaginous degeneration of the mitral valve and mitral stenosis—[A 68 year old woman was seen in 1822 with a totally irregular pulse] The beats of the ventricle are irregular and intermittent these intermittences in general are preceded by two quick rapid contractions which are closely

[a] This reference appears to contradict the statement of Corrigan (298) that Laennec looked on murmurs and thrills as unrelated phenomena

He expounded the important concept that gallop sounds are an exaggeration of normal elements 'If one auscultates a goodly number of healthy persons, one will not be long in discovering that diastole is not always absolutely silent, that in the part of the cardiac cycle where the abnormal sound responsible for the gallop is located, there is sometimes already, in vestigial form, something which, where exaggerated, could become the sound in question' Dock (357, p 36) wrote as follows

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[7] Translation by Mounsey (1122)

knew little of the patient's subsequent course until eleven years later, 1872, when he received by mail from the patient's physician the heart of Patrick M., who had recently died of acute pneumonia. The heart was normal in size and healthy in appearance. Much was Gairdner's surprise when he opened the heart in the presence of his students and found a pedunculated ball valve tumor of the right atrium. The heart valves were completely healthy. Gairdner recognized this case as convincing evidence for the auricular systolic origin of the presystolic murmur. However, Brockbank dismissed this evidence with the suggestion that the tumor held open the valve and permitted regurgitation around it.

Edward Mansfield Brockbank (1866–) of Manchester, England, beginning in the last part of the last century (180, 183), has been defending the protosystolic nature of the presystolic murmur. In 1957 in a letter to me Brockbank stated that he had seen no reason to change his view. In the 1920 (11th) edition of his small book on *Heart Disease* (183) he emphasized the following as important features of the presystolic murmur: it rises in pitch progressively with its increase in vigour, and is at its highest pitch and maximum force when it is abruptly terminated by the accentuated first sound (p. 63).

He continues: How the rising pitch character of the murmur is imparted to it during its (atrial systole) has never been explained. This must be produced at an orifice which progressively closes whilst the murmur is being produced at it (p. 64-65).

Figure 9 is a copy of the illustration Brockbank[a] used to clarify the mechanism he defended. He compared it to the operation of the lips in producing the sound as the lips come closer together to produce the rising pitch. See footnote on p. 23 for a discussion of the status of this controversy in recent years. Despite the advent of phonocardiography, which permits

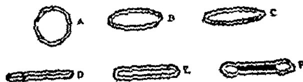


Fig. 9. Brockbank's conception of the genesis of the presystolic murmur of mitral stenosis (183).

accurate timing of the presystolic murmur, advocates for its protosystolic nature remain.

The *bruit de rappel* in mitral stenosis was interpreted as representing asynchronous closure of the arterial valves until the 1870's and early 1880's, when *Paul Guttmann*[b] (1834–1893) in Berlin (618) and *Arthur Ernest Sansom* (1838–1907) at the London Hospital (1333) supported the view that the second element of the double sound proceeds from opening of the diseased mitral valve. The theory was formalized and the sound named in his doctorate thesis of 1888 (1310) by *R. J. M. Rouchès*[c], a student of Potain, who is said to have suggested the designation *claquement d'ouverture de la mitrale*. Thayer (p. 23) suggested the English equivalent *mitral opening snap*; the phenomenon had been known by earlier English writers and some later ones as the double shock sound. It is mainly since 1948 and the advent of mitral valve surgery that physicians have appreciated the value of the opening snap in differentiating predominant mitral stenosis from predominant mitral regurgitation.

Sir *James Mackenzie* (1011) described and correctly explained the disappearance of the presystolic murmur with the development of

[a] Brockbank's other interests have included medical history (the biography (1944) of his fellow Mancunian John Dalton is noteworthy), mule spinner's cancer (a cutaneous affliction of cotton mill workers of Manchester), general pediatrics (*Children: Their Care and Management*, 1911), and life insurance examination (*The Conduct of Life Assurance Examinations*, 1912).

[b] Guttmann in 1869 (620) wrote also about the split heart sound of mitral stenosis. He had apparently not yet developed the concept of the mitral opening snap.

[c] With total irregularity of the heart Rouchès said the opening snap was more striking after long diastolic periods. He found it loudest over the area of the mitral valve (medial from the apex). He claimed it came and went. A defect of his analysis was the lack of autopsy to be sure that the emphasis on splitting of the second sound as a characteristic and frequent phenomenon in mitral stenosis. In this he was in error. Rouchès was born September 6, 1864, at ... Department of Cantal in southern France. I have no information on his career after 1888.

spaced The contractions of the left ventricle have a very forceful impulse and rather a ringing sound those of the auricles are accompanied by a murmur comparable to the blowing of a bellows, or even more to the placental murmur, the hand applied to the precordial region senses a vibratory motion deep but very marked it is abruptly replaced by the ventricular movement

It is clear that what Bertin heard was an unusually prolonged 'passive' diastolic murmur of mitral stenosis In stating that it occurred with auricular systole he was following the authority of Laennec that the second heart sound is related to atrial systole This view was not challenged until a few years later Furthermore the total irregularity of the heart beat indicates that atrial fibrillation was probably present and a true 'auricular systolic' murmur impossible He also described the snapping first sound the diastolic thrill and possibly the opening snap following the second sound ('two quick rapid contractions')

The presystolic murmur was described and named by *Sulpice Antoine Fauvel* (1813–1884) *chef de clinique* at Hotel Dieu Paris in 1843 (455) 'I conclude from the facts stated in this memoir that a morbid presystolic bruit localized at the apex of the heart is in the existing state of science the stethoscopic sign which points with the greatest probability to contraction of the mitral orifice' In a footnote referred to the term presystolic he stated further 'I borrow the expression from M Gendrin (540) while admitting that he used it in a different sense' Fauvel later became one of the greatest of modern sanitarians He did work in public health in Turkey for 19 years and was later General Inspector of Health Services of France (454) This was a sharp departure from his clinical contributions as a young resident physician (Fig 7)

At approximately this same time *Skoda* pointed to enfeebled aortic sounds and accentuated pulmonary second sound ('Skoda's sign') as an important clue to the diagnosis of mitral stenosis and *Bouillaud* related his *bruit de rappel* to this lesion But most including Bouillaud, Fauvel's chief at the Hotel Dieu refused to accept Fauvel's description of the presystolic murmur

In 1861 *William Tennant Gairdner* (1824–1907) then of Edinburgh, later Regius Professor of Medicine in Glasgow (541), also described the presystolic murmur and suggested the designation "auricular systolic" (506) At about the same time *Duroziez* (392) invented his onomatopoetic device 'fout ta ta rou" and gave lucid descriptions of 'pure' mitral stenosis which resulted in the condition being known as Duroziez's disease in the French literature

Finally, not long after 1871, when the convincing paper of *C Hilton Fagge* (1838–1883) of Guy's Hospital, London, appeared (448), the presystolic murmur was accepted as the leading auscultatory sign of mitral stenosis Fagge (448) quotes Hyde Salter (1823–1871), also of Guy's as stating in a students lecture in 1869 that 'now anyone who should fail to recognize and identify this sound [presystolic murmur] would not only be unfit to hold the place of an accomplished and critical physician but could hardly be considered as a decently informed member of our profession'

Although the descriptive aspect of the 'presystolic murmur' and its association with mitral stenosis were well established a controversy long continued as to the genesis of the murmur Gairdner's 'auricular systolic' concept was not accepted by all An alternative interpretation which has been defended to the very present (see p 295) was that the murmur is actually in early systole and is produced by regurgitation at the mitral valve

Gairdner (505, 541) reported on a case illuminating in connection with the origin of the presystolic murmur In 1861 he first saw an Irish labourer Patrick M , aet about 20 'He suffer[ed] no very great amount of inconvenience from his disease except from a remarkable undulating movement in his neck for which he came over to Edinburgh [from Dundee] about two years ago to consult Mr Syme supposing that it was something that might be cured by surgery' Gairdner found that the pulsation was venous and that there was an extraordinarily loud precordial murmur which resulted in the patient being long a 'showcase' 'The cardiac murmur begins immediately after the second sound continues *diminuendo* throughout the pause and then goes on *crescendo* up to the first sound at which it stops abruptly' Gairdner

(third left intercostal space at the sternal margin) presumably derives its name from *Wilhelm Herman Erb* (1840–1921). I have, however, been unable to find definite confirmation of this. Erb (1491) is better known as a neurologist. Erb's spastic paraplegia comes immediately to mind.

Austin Flint (1812–1886) was a peripatetic American professor of medicine who was born in Petersham, Massachusetts, but lived and taught in Buffalo, Louisville, New Orleans, Brooklyn, and other places (Fig. 10). He was in New York in 1862 when he reported on the occurrence of a presystolic murmur simulating that of mitral stenosis in patients with aortic regurgitation who, however, were found at autopsy to have no lesion of the mitral valve (464). In 1859 Flint won the first prize offered by the American Medical Association for an essay on the subject of "The Clinical Study of the Heart in Health and Disease." Flint wrote a textbook of medicine and one of physical diagnosis.

Balthazar Foster (1840–1913) claimed that it is possible to predict from the radiation of the murmur of aortic regurgitation which aortic cusp is predominantly damaged (471). This was the basis of Foster's rule and of the Balthazar Foster murmur (470). Some recent observers (782) have been unable to confirm this impression, although others (652, 1200) support it in modified form.

In 1900 *George A. Gibson* (1854–1913), professor of medicine in Edinburgh, first described clearly the characteristic continuous murmur of patent ductus arteriosus (546). In England and to some extent in America this is known as the Gibson murmur. "At the base of the heart, an almost continuous rushing murmur is heard, beginning shortly after the first sound, which is quite clear at its commencement, and continuing almost to the next first sound. It is best heard in the third left interspace about $2\frac{1}{2}$ inches from the midsternum, but is heard all over the base of the heart. The second sound is heard distinctly in the middle of this murmur; it is distinctly accentuated and sometimes doubled" (546).

In 1907, at the same time as Hirschfelder and Thayer in Baltimore and shortly after Obraztsow in Kiev, Gibson (547) discovered the normal third heart sound and suggested to Einthoven that he seek it in his string galvanometer phonocardiogram (416).

See p. 18 for the history of the Hodgkin-Key murmur.

In 1905 *Nikolai Sergeyevich Korotkoff* (1874–1920) of St. Petersburg, Russia, described the arterial sounds useful in estimating blood pressure (815). He was a privat dozent in the Imperial Military Medical Academy, a first-rate medical school graced by Pavlov as Professor of Physiology. His succinct description of the blood pressure sounds was only reported stenographically (Fig. 11). For translations see Lewis (906) or Pickering (1208). Apparently Korotkoff had been a surgeon in the Russo-Japanese War. Following the recommendation of Pirogoff, famous Russian surgeon, he had used auscultation in the differentiation of solid tumors from peripheral aneurysms (284). From this he was led to a study of normal arterial sounds.

Jacob Lerman (1902–) and *James H. Means* (1885–) of the Massachusetts General

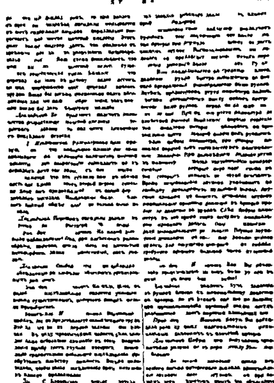


Fig. 11. Korotkoff's description of the blood pressure sounds. (Courtesy of New York Public Library.)

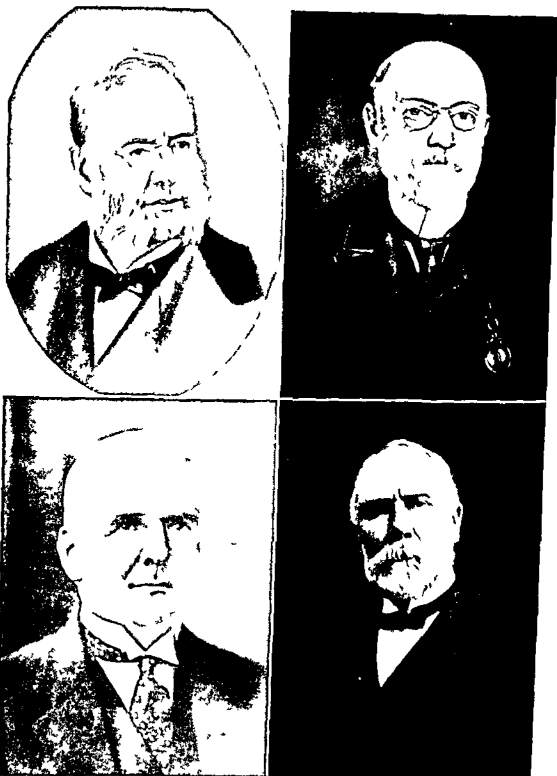


FIG 10 (*Upper left*) Austin Flint (*upper right*) Graham Steell (Courtesy of Dr A Morgan Jones Manchester) (*lower left*) Carey Coombs (Courtesy of Dr John Co[illegible]h Bristol) (*lower right*) James Mackenzie

the back pursued them into the neck and even into the thigh (663) And he said of himself 'As long as my heart beats, I shall listen to the heart of others' (1568)

Among the many auscultatory signs which Duroziez described is the waxing and waning of the diastolic murmur of tricuspid stenosis with inspiration and expiration respectively (405)

Erb's area (or point) the usual area of maximal audibility of the murmur of aortic regurgitation

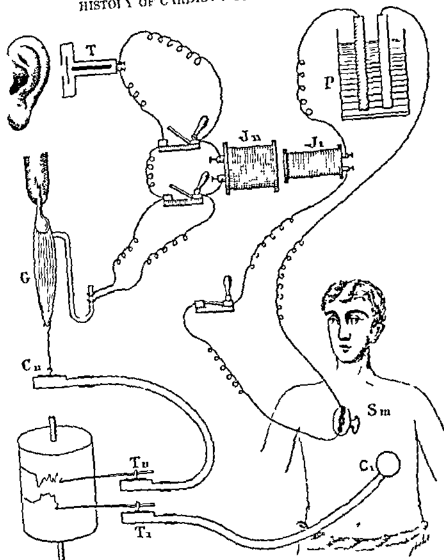


Fig 12 Huerthle's apparatus using frog nerve muscle preparation for registering heart sounds (1893)

ductorium the secondary coil of which was connected to the nerve of a frog nerve muscle preparation which marked on a smoked drum by means of an attached lever (Fig 12)

The first phonocardiograms in anything approaching the modern sense were made by Willem Einthoven (with Geluk) in Leyden in 1894 (418) using Lippman's capillary electrometer (Fig 13)—with which the first electrocardiograms had also been made

Direct phonocardiograms (Fig 14) that is recordings of the precordial vibrations with optical amplification were first made (478) by Otto Frank (1865–1944) of Munich in 1904 (1533) This method making use of the so called Frank segment capsule, was modified and improved on by many workers among them Carl J Wiggers (1545 1548 1554 1555) in Cleveland, Ohio, and Orias and Braun Menendez in Argentina (1166) Using the direct method O Weiss wrote an early (1909) monograph on phonocardiography in which many phenomena—e g arteriovenous fistula splitting of heart sounds stenosis and regurgitation at the aortic and mitral valves—were displayed graphically for the first time and the term "phonocardiogram" used The method suffered from limitations in the frequency response of the membrane used in the

Hospital, Boston, described (881) a scratchy systolic murmur in a high proportion of cases of severe thyrotoxicosis. Often the superficial and scratchy character is so pronounced as to suggest a pericardial friction rub. This is the so called *Lerman Means scratch* which is thought to be caused by high flow, in terms of both velocity and volume, through the pulmonary artery. As usual, earlier descriptions of the phenomenon can be found, for example in 1920 Goodall (567) wrote as follows: "A superficial pericardial rub is often heard, this is most common over the pulmonary base ... It is probably produced mechanically."

In 1879 *Henri Roger* (1811–1891) of Paris described (1304) the long systolic murmur of uncomplicated interventricular septal defect, the *Roger murmur*. In 1841 Barth and Roger had first published a textbook on auscultation, which went through many editions.

Skoda's sign in mitral stenosis is referred to on p. 22 and p. 24.

In an article entitled "the murmur of high pressure in the pulmonary artery" *Graham Steell*[14] (1851–1942) of Manchester, England (Fig. 10) described the murmur which bears his name (1436). Although this pulmonary diastolic murmur, which is often difficult to differentiate from an aortic diastolic murmur, is usually associated in our thinking with mitral stenosis and probably indeed occurs most often in this situation, it can, as indicated by Steell, occur with any type of pulmonary hypertension.

I wish to plead for the admission among the recognized auscultatory signs of disease of a murmur due to pulmonary regurgitation, such regurgitation occurring independently of disease or deformity of the valves, and as the result of long continued excess of blood pressure in the pulmonary artery.

In cases of mitral obstruction there is occasionally heard over the pulmonary area (the sternal extremity of the third left costal cartilage), and below this region for the distance of an inch or two along the left border of the sternum, and rarely over the lowest part of the bone itself, a soft blowing diastolic murmur immediately following, or, more exactly, running off from the accentuated second sound, while the usual indications of aortic regurgitation, afforded by the pulse, etc., are absent. The maximum intensity of the murmur may be regarded as situated at the sternal end of the third and fourth intercostal spaces. When the second sound is reduplicated the murmur proceeds from its latter part. That such a murmur as I have described does exist there can, I think, be no doubt. ... The murmur of high pressure in the pulmonary artery is not peculiar to mitral stenosis, although it is most commonly met with as a consequence of this lesion. Any long continued obstruction in the pulmonary circulation may produce it. The pulmonary valves, like the aortic, do not readily become incompetent, apart from structural changes. Probably no amount of blood pressure in the pulmonary artery will render them so suddenly as at least theoretically the mitral valves may be rendered incompetent. Changes in the vessel with widening of its channel and eventually, of its orifice, long precede the occurrence of incompetence of its valves. The pulmonary murmur of high pressure is probably never persistent at first, and one of its most remarkable features is, as a rule, its variableness in intensity. On some days it will be distinctly heard, on others it will be indistinct or even inaudible, while extreme accentuation of the pulmonary second sound is always present, the closure of the pulmonary semilunar valves being generally perceptible to the hand placed over the pulmonary area as a sharp thud. An accentuated second sound is no way incompatible with a certain amount of incompetence of the semilunar valve; on the contrary, an accentuated second sound is associated with a regurgitant murmur as clinically common.

In 1891 Pawinsky (1192) presented necropsy evidence in support of Graham Steell's view. It is probable that the Graham Steell murmur was described by Hope in 1832 (p. 18); what had been known for some time as "Hope's early diastolic murmur" was probably the same as what we now call the Graham Steell murmur.

Ludwig Traube (1818–1876) of Berlin described (1474) a double sound heard over peripheral arteries in aortic regurgitation without pressure with the stethoscope. He found this sign also in cases of typhoid fever. He also made early studies of what he called *Galopprythmus* (1474).

THE AGE OF PHONOCARDIOGRAPHY

An early (1893) method for making the heart sounds inscribe their own record was that used by K. Huerthle of Breslau (720, 721). He led the output of the microphone through an in-

[14] Steell (153) was the son of Sir John Steell, the sculptor who designed the familiar monument to Sir Walter Scott in Princes Street, Edinburgh. He was a close friend of Mackenzie (see p. 20) who while in practice in Burnley, near Manchester, frequently made hospital rounds with him.

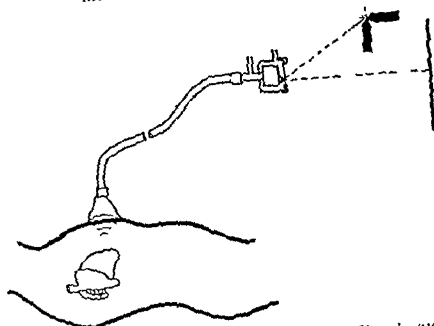


Fig 14 Schema of direct phonocardiography (From Orias and Braun Menendez (1166))

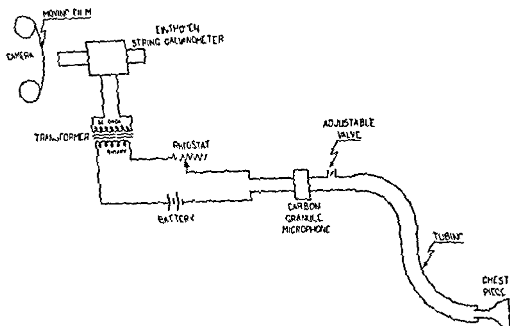


Fig 15 Schema of Einthoven's string galvanometer phonocardiograph (124)

now a generation of Heart Station *habitués* totally unfamiliar with the string galvanometer just as the preceding generation was unfamiliar with the capillary electrometer

In direct phonocardiographs and in phonocardiography using the string galvanometer since no amplifier was used and no electrical means for filtration or selective amplification were available it was necessary to use acoustical filtration to get rid of the low frequency vibrations of very large excursion which obscure the much fainter vibrations in the auscultatory range of

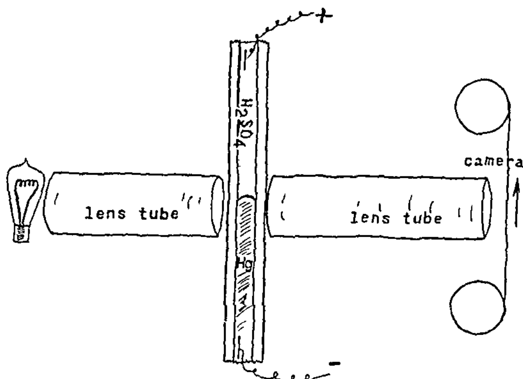


FIG 13A Schema of Lippmann capillary electrometer (Adapted from Fig 202 p 643 W M Bayliss *Principles of General Physiology* London Longmans Green 1915 (70))

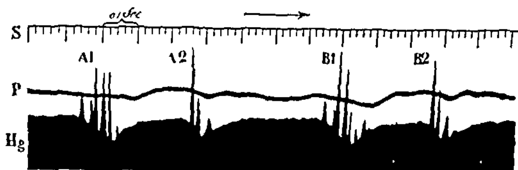


FIG 13B Phonocardiograms made with capillary electrometer (From Einthoven and Geluk (1894))

segment capsule Many of the phonocardiograms made with this direct method have an appearance suggesting that the undamped vibrations of the recording membrane were excited in a non specific manner In one publication (146) in connection with diastolic sounds demonstrated by this method *it was stated, since the ribs* intercostal spaces and sternum and indeed the whole body receive an impact during atrial systole and during rapid ventricular filling, the possibility is not eliminated that the capsules might have set their highly undamped membranes into oscillation

In 1907 (415) Einthoven[16] (1860–1927) who in 1924 was awarded the Nobel prize principally for his work in electrocardiography reported on the use of the string galvanometer for recording the heart sounds (Fig 15) Although this method was used by Sir Thomas Lewis (902 904) by Battaerd (61) a pupil of Einthoven and others (311, 312) including in recent times Dock and Rytand the tremendous range of intensities encountered in cardiovascular sound far exceeding anything in the electrocardiogram led to frequent accidents with fracture or other damage to the string Nonetheless much valuable information was uncovered It is startling to consider that with the development of electronic amplification making possible use of various galvanometers such as the d'Arsonval and the advent of the cathode ray oscilloscope there is

[16] For an excellent photograph of Einthoven with his string galvanometer see the frontispiece of Burch and Winsor's *Primer of Electrocardiography*

the inverse relationship between the duration of the interval between the second heart sound and the mitral opening snap and grade of mitral obstruction. These two features have been used in the last few years in the quantitative assessment of mitral stenosis (1520) (778).

The appearance of several monographs have been landmarks in the history of phonocardiography and of cardiovascular sound in general.[1]

1 Orias, O. and Braun Menendez, E. (From Buenos Aires.) *The Heart Sounds in Normal and Pathological Conditions*. London, Oxford University Press, 1939.
2 Lian, C., Minot, G. and Welti, J. J. [From Paris.] *Phonocardiographie. Auscultation collective.* (Acoustique Technique Clinique.) Paris, Masson, 1941.
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5 Calo, A. [From Turin.] *Les bruits du coeur et des vaisseaux*. Paris, Masson, 1950.
6 Schmidt-Voigt, J. *Herzschalldiagnostik in Klinik und Praxis*. Stuttgart, Georg Thieme, 1951.
7 Holldack, K. and Wolf, D. [From Heidelberg.] *Atlas und kurzgefasstes Lehrbuch der Phonokardiographie*. Stuttgart, Georg Thieme, 1956.
8 Weber, A. *Atlas der Phonokardiographie. Optische und magnetische Niederschrift des Herzschalls ugleich 2. Auflage der Herschallregistrierung* (ref. #3). Darmstadt, Dietrich Steinkopff, 1956.

In addition Mannheimer of Stockholm produced a monograph-length opus on calibrated phonocardiography which was published as a supplement to Acta pediatrica Scandinavica (1031). Butterworth of New York City (204) and Rappaport of Denver (1240A) have published monographs on cardiac auscultation and used phonocardiograms for illustrative purposes.

THE STRUCTURE AND FUNCTION OF THE HEART VALVES IN HEALTH AND DISEASE

So intimately is cardiovascular sound related to the heart valves that some survey of the development of knowledge about the anatomy, physiology and pathology of these structures is indicated. However, so extensive is this subject that only a brief review is possible.

STRUCTURE. According to Galen (the original manuscript is lost) Erasistratus, who lived about 250 years B.C., was familiar with the heart valves and named the atrioventricular valves *tricuspid* and the arterial valves *sigmoid* (509). The early Greek letter sigma, unlike the modern one, was shaped like our letter C. The cup-like shape of the cusps of the arterial valves is well indicated by the designation.

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[16] The quotation above is provided by Davison (340). I have been unable to locate it in the translation of Benjamin Alexander published in 1769.

frequency. Acoustical filtration was accomplished by means of an air leak in the tube connecting the chest piece with the Frank capsule or microphone.

The advent of the vacuum tube and electronic amplification produced a change in the technique of phonocardiography in essentially all parts of the world. The review of Rappaport and Sprague (1244) in 1942 marked the end of any other methods of phonocardiography and outlined the use of electronic amplification and galvanometers of various types. Maurice B. Rappaport, Howard B. Sprague and their collaborators, of Boston have emphasized the separate areas of usefulness of what they term stethoscopic and logarithmic phonocardiograms (p. 79).

All phonocardiography hitherto discussed is oscillographic phonocardiography. A major departure was represented by adaptation of the Bell Telephone Laboratories' method of sound spectrography to the study of heart sounds and murmurs, by Geckeler and colleagues (532) who called it cardiospectrography, and by my colleagues and me (1085) who called the method *spectral phonocardiography*. Spectral phonocardiography is a development of this decade.

The following is a partial enumeration of some of the contributions made by phonocardiographic means or with phonocardiographic confirmation.

Einthoven (416) published the first recordings of the physiologic third heart sound.

In 1914 *C. J. Benjamins* (84), an ear nose and throat doctor in Utrecht, demonstrated that an atrial sound can be recorded from the esophagus in all cases of sinus rhythm. In the same year *Eveleth W. Bridgman* (1888–1958) of Baltimore, published recordings of the atrial heart sound in normal boys (175).

In 1915 *Sir Thomas Lewis* (1881–1945) of London provided phonocardiographic confirmation for Mackenzie's observation that the presystolic murmur of mitral stenosis disappears with the advent of atrial fibrillation (904). He provided one of the first published recordings of a musical aortic diastolic murmur of retroverted aortic cusp (902, Fig. 18).

In 1918, *Frank N. Wilson* (1890–1952), later of Ann Arbor, with Jamieson presented satisfactory phonocardiograms of three cases of musical aortic diastolic murmur (1570). He noted the decrescendo or crescendo-decrescendo pattern of intensity. He noted that there was also a decrescendo in frequency. In his three cases, the frequency at the beginning was estimated at 150, 140, and 170 c.p.s., and at the end at 125, 115, and 140, respectively.

In 1915 *Battaerd*, student of Einthoven, recorded atrial heart sounds in atrial flutter with heart block (61). He also studied extrasystoles.

Charles C. Wolferth (1887–) of Philadelphia, working for the most part with *Alexander Margolies* (1894–), reported studies which did much to familiarize the English-speaking world with the mitral opening snap (1040), pointed out the relationship of the intensity of the first heart sound to the PR interval of the electrocardiogram (1578), introduced the now generally accepted concept of summation gallop (1577). In 1935 from an analysis of the heart sounds in bundle branch block (1576), Wolferth concluded that the then generally held view that the right bundle was blocked in cases with the electrocardiographic pattern of so-called common bundle branch block, was false and that in fact there was block of the left bundle—an unusual triumph for phonocardiography.

Camille Lian (1882–) of Paris, one of the leading phonocardiographers, has with various collaborators made many contributions to the precise description of cardiovascular sound: Pleuropericardial systolic clicks (920), the murmur of patent ductus arteriosus (935), the early diastolic snap of pericardial constriction (914), the early systolic click of disease of the aorta or pulmonary artery (939), musical extracardiac murmurs (913).

In 1937 in a beautiful article based on fourteen patients *Routier* (1316) demonstrated the pattern *of the continuous murmur in patent ductus arteriosus*.

In 1941 *Weiss and Joachim* (1522) pointed out that in mitral stenosis the first heart sound is delayed in relation to the QRS complex of the electrocardiogram and as compared with the situation in normal subjects. The phenomenon was rediscovered by *Cossio and Berconsky* (300) in Argentina in 1943 and by *Howard B. Sprague* and collaborators (1097) in Boston in 1951. Sprague and co-workers (1097) also pointed out

the inverse relationship between the duration of the interval between the second heart sound and the mitral opening snap and grade of mitral obstruction. These two features have been used in the last few years in the quantitative assessment of mitral stenosis (1525) (778).

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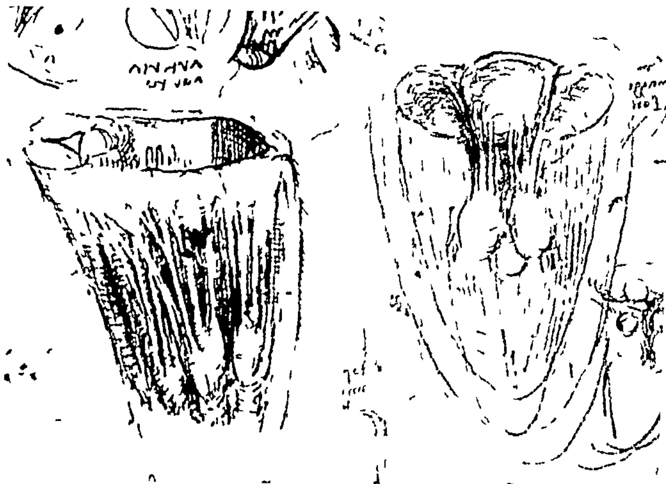


FIG 16 Drawings of the functional anatomy of the heart by Leonardo da Vinci A (left) Tricuspid valve op[en] B (right) Tricuspid valve closed (From O'Malley and Saunders (1158).)

vessels that the movement and flow of the blood are temporarily arrested

In 1840 Kurschner (829) described small myocardial fibers in the atrioventricular valves. These fibers are basically atrial in origin and have been suggested as a contributing factor in valve closure, a dubious theory.

Diseased valves, particularly mitral and aortic, were described by John Mayow (1643–1670) of Oxford (1054), Théophile Bonet (1620–1689) of Geneva, Vieussens (1641–1715) of France, Lancisi (1654–1720) of Italy (1594), Albrecht von Haller (1708–1777) of Switzerland, Jean Baptiste Senac (1693–1770) of France, Giovanni Morgagni (1682–1771) of Padua, Matthew Baillie (1761–1823) and William Cowper (1666–1709) of England (313), and others before Laennec.

FUNCTION. The functional anatomy of the heart valves fascinated Leonardo da Vinci (1452–1519), who left notes and drawings (Fig 16) which are now the property of the British royal family and are housed in the library at Windsor Castl[e] (1158). In general he had a clear notion of th[e] anatomy of the valves and portrayed their struc[-]ture in a much clearer manner than did, fo[r] example, Vesalius' illustrator Calcar. Specifically, at least two functional observations were made: (1) By analysis of the geometry, Leonardo estab[-]lished that a three-cusp arterial valve provide[s] maximal strength in the closed position and minimal obstruction to forward flow when open. (In 1936 in a clinicopathologic report Ku[...] (798) demonstrated that a quadricuspid pul[-]monary valve is prone to regurgitation.) Further[-]more, a bicuspid valve produces mild obstruction to forward flow such that a murmur may be pro[-]duced and atherosclerosis and bacterial endo[-]carditis develop secondary to the locally altered pattern of flow. In 1873 Longworth[17] (963) Pro[...]

[17] Longworth's reasoning was somewhat different. He concluded that in order to open (without obstruc[-]tion to flow) the sum of the lengths of the free borders of the cusps should equal the circumference of the aortic

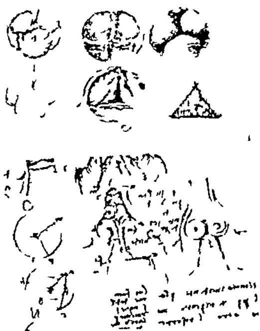


FIG 16 *C Above* Semilunar valve open and closed (several views). Note triangular shape of orifice when valve is open *D Below* Vortex formation behind cusp and hypothetical role of same in closure of semilunar valves (From O'Malley and Saunders (118).)

fessor of Anatomy in the Medical College of Ohio Cincinnati came independently to the same conclusion as Leonardo about the superiority of a tricuspid arterial valve (2) Leonardo conceived of vortices behind the semilunar cusps being responsible for a partially closed position of the cusps during ventricular ejection and for subsequent closure of the valve The same idea—that eddies behind the valves open up like tightly wound watch springs and close the valve when ventricular ejection ceases—was expressed by Krehl in 1891 The existence of vortices of the type drawn by Leonardo in the sinuses of Valsalva during ventricular ejection is theoretically likely on hydrodynamic ground Such vortices may be important in the formation of the sinuses of Valsalva an illustration of the role of hemodynamic factors in the morphogenesis of the

ring and the length of the free borders of one cusp should equal the diameter By means of a model of paper he illustrated to his classes that a tricuspid valve most nearly satisfies these requirements The lengths of the free borders of the three cusps approximate the circumference of the circle as 3 approximates 3 1416 (value of π)

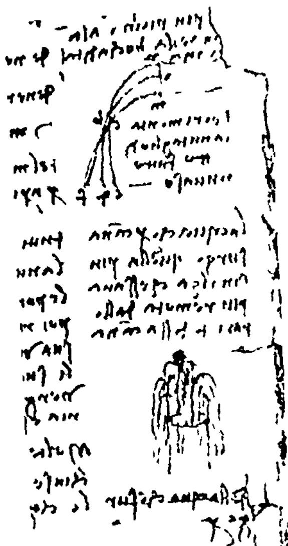


FIG 16 *E* Leonardo visualized the formation of vortices by a method analogous to the way the water falls in flowing from a horizontal or upright tube (From O'Malley and Saunders (118).)

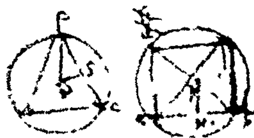


FIG 16 *F* Drawing to indicate that the quadricuspid valve is weaker because of greater height of the triangle (as drawn here by Leonardo) (From O'Malley and Saunders (118).)

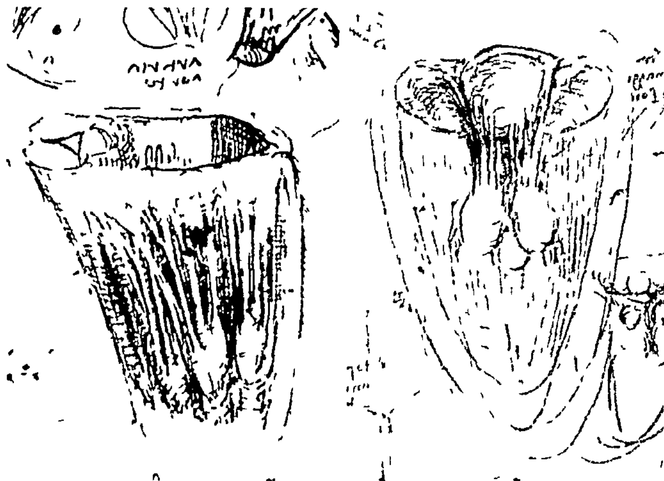


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vessels that the movement and flow of the blood are temporarily arrested.

In 1840 Kurschner (829) described small myocardial fibers in the atrioventricular valves. These fibers are basically atrial in origin and have been suggested as a contributing factor in valve closure, a dubious theory.

Diseased valves, particularly mitral and aortic, were described by John Mayow (1643–1670) of Oxford (1054), Théophile Bonet (1620–1689) of Geneva, Vieussens (1641–1715) of France, Lancisi (1654–1720) of Italy (1594), Albrecht von Haller (1708–1777) of Switzerland, Jean Baptiste Senac (1693–1770) of France, Giovanni Morgagni (1682–1771) of Padua, Matthew Baillie (1761–1823) and William Cowper (1666–1709) of England (313), and others before Laennec.

FUNCTION. The functional anatomy of the heart valves fascinated Leonardo da Vinci (1452–1519) who left notes and drawings (Fig 16) which are now the property of the British royal family and are housed in the library at Windsor Castle (1158). In general he had a clear notion of the anatomy of the valves and portrayed their structure in a much clearer manner than did, for example, Vesalius' illustrator Calcar. Specifically, at least two functional observations were made. (1) By analysis of the geometry Leonardo established that a three cusp arterial valve provides maximal strength in the closed position and minimal obstruction to forward flow when open. (In 1936 in a clinicopathologic report Kissin (798) demonstrated that a quadricuspid pulmonary valve is prone to regurgitation.) Furthermore a bicuspid valve produces mild obstruction to forward flow such that a murmur may be produced and atherosclerosis and bacterial endocarditis develop secondary to the locally altered pattern of flow. In 1873 Longworth[17] (903) Pro

[17] Longworth's reasoning was somewhat different. He concluded that in order to open (without obstruction to flow) the sum of the lengths of the free borders of the cusps should equal the circumference of the aortic

Carswell (1793–1857) (240) Marc d'Espine (1037) Carswell came to this conclusion from examining on the service of Louis in Paris a patient with an aneurysm of the ascending aorta presenting to the right of the sternum Over the body of the heart the first and second sounds were faint (making impact of the heart against the chest wall unlikely as a mechanism) but over the tumor the second sound was maximal and occurred as the systolic swelling of the tumor subsided (140 p 151) But none established the origin of the second sound more convincingly than did *Joseph Rouanet* (1797–1865) of Paris in his thesis for the M D degree (1832) He affixed a bladder in the outflow tract of the ventricle in such a manner that he could simulate ventricular systole by squeezing it Then listening over the arterial valve he heard a sound like the second heart sound when he relaxed pressure on the bladder (Fig 17 1)

Billing (103) had attributed both heart sounds to tensing of valve curtains the atrioventricular valves in the case of the first sound the arterial valve in the case of the second But it was almost exclusively intuition that led him to this conclusion On the other hand Rouanet (1314) who likewise held that both sounds were due to tension of the valves as much as or more than the actual collision of the cusp margins provided experimental evidence for this view Rouanet stated at the beginning of his thesis that he would cover the following topic

(1) I will briefly review what has been published on this subject (2) I will show that action of the heart valves is accompanied by sound (3) that these sounds coincide with those of the heart and that in granting that these are the same one provides perfect explanation for the observed fact (4) that no existing theory is admissible (5) I shall end with certain consideration of the orifices and valves of the heart and abnormal sound

Rouanet's experiments consisted of (1) the use of the model (Fig 17 1) described in the text below in which arterial valve action was simulated (2) the use of a second type of model in which action of both the mitral and the aortic valve was simulated and (3) the tensing of membranes and relating of the character of the sound produced to the physical structure of the membrane (The second type of experiment was described in his second publication in 1844 (417))

I put the part below the sigmoid valves around a glass tube about an inch in diameter and 2 or 3 inches long joining below a bladder similarly fixed to it and full of water The portion of the vessel above the valve was fixed to the lower end of a second tube of the same diameter and more than four feet in height in order to compensate by the elevation of the liquid column for the forces of impulsion which normally exist be it on the part of the blood or of the arteries and the neighboring part Then seizing the apparatus at the level of the valves which remain free and applying my ear in such a manner that it is separated only by the fingers I would squeeze the bladder suddenly with the other hand I would imitate insofar as possible the beating of the heart as to the quantity of liquid which I made pass into the upper tube with each beat and as to the intermittence of the action which I exer

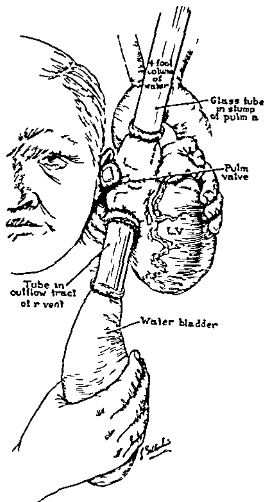


Fig 17 1 Rouanet's experiment (see text)

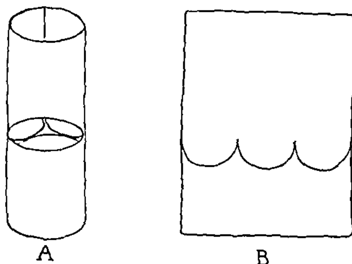


Fig 17 Longworth's model (see text)

cardiovascular system The partially closed or at least mid position of the valves during ventricular ejection is now well established Although it is of interest how far the genius of Leonardo was able to penetrate into the mysteries of valve function, historically his studies were of no significance because they were completely unknown until relatively recent times

It has probably been evident since Harvey at least that the closure and opening of valves is fundamentally the result of a change in polarity of the pressure differential on the two sides of the valve The details of the mechanism of valve closure were filled in later however The ancillary or contributory mechanisms account particularly for the fact that the valves close without attendant regurgitation when these mechanisms go awry, regurgitation is likely to develop

In the instance of the semilunar valves Ceradini (248) established in 1873 that the valve assumes an intermediate position of partial closure during ventricular ejection He suggested that at the end of ejection the cusps are pulled together in the wake of the systolic ejection jet and that the margins of the cusps are virtually in contact at the time that the polarity of pressure differential is reversed

A similar mechanism in the closure of the atrioventricular valves was supported by the work of Lucciani about 1903, Yandell Henderson (670) with Johnson about 1912, and Dean about 1916 (341) Henderson in particular emphasized the importance of atrial systole in assisting closure of the atrioventricular valves, by a breaking of the jet" mechanism comparable to that in the case of the arterial valves (*cf* p 112)

Beginning with Rouanet, who reported in 1832 (1314), and Baumgarten in 1843 (63), various authors have built systems to simulate the pumping action of the heart and study valve action An interesting example is the system used by Gad and illustrated in his publication (502) in 1886 In recent years Ian McMillan (1088 1089) of London adapted this method, combined with cinematography, for the study of diseased valves in human hearts and others (331, 803) have followed suit Cinematographic studies of heart valves have been performed (1407) in excised, surviving, saline perfused hearts of animals In intact animals Rushmer had made x ray cinematographic investigations of the movement of heart valves—to which radiopaque markers have been affixed (1326)

THE PHYSICS AND PHYSIOLOGY OF CARDIOVASCULAR SOUND

THE HEART SOUNDS The error of Laennec's view that the first and second sounds are due to ventricular and atrial systole respectively was first pointed out in 1829 by *John William Turner* (1790–1836) Professor of Surgery (!) in Edinburgh who had only to recall that Senac Lancisi Haller and a number of others knew that the atrium contracts immediately before the ventricle not after the ventricle (1477) One of his students described Turner as a timid shy man who could not look his class in the face and seemed fitted by nature for anything rather than the duties and responsibilities of an operating surgeon (588) Perhaps he was particularly fitted by nature for challenging Laennec's doctrine! On his premature death Turner was succeeded by the famous Sir Charles Bell

Although other theories continued to be embraced by some workers for several decades (see below) shortly after 1830 the second sound was related to closure of the arterial valves by several writers among them Billing[18] Robert

[18] The prize of the century for comprehensive titles is certainly due Billing's *On the auscultation and treatment of the affection of the heart*—all in an article three and one half pages in length!

and instantaneous force we are obliged to agree that it should produce a sound perceptible to the ear.

Rouanet related the difference in character of the two heart sounds to physical difference in the heart valves. The first sound is loud and depends to a certain extent on the energy of the ventricle and is duller than the second. The valves which occasion it are larger and the walls which conduct it thicker. The second sound is sharper because the valves are smaller, thinner and attached to more sonorous walls.

Rouanet is of some special interest to American cardiologists because he spent the last eight years of his life as a practitioner in New Orleans. Born in southern France in 1797, the son of an illiterate peasant, Rouanet (213, 344, 447, 1075) was befriended by the proprietor of the estate where his father worked. He was educated for the priesthood, being sent to seminary in Paris about 1820. However, he soon renounced his ecclesiastical intentions and undertook to earn a livelihood by teaching Latin and Greek. His formal medical education was between 1828 and 1832. From the time of qualification until 1847 he practiced medicine in Paris. Because of failure to enlarge the circle of his clientele and also because of heavy losses in stock market peculation, he emigrated to New Orleans in 1847. There he appears to have been an important addition to the medical community. An entertaining episode is described by a colleague and friend (447):

> One day a young colleague wished to have a joke at Rouanet's expense. He brought to Rouanet's house very gravely an anatomical specimen saying it was an infant's heart which showed most singular anomalies. It seems that Rouanet studied them with much curiosity. But it was the heart of a goose! I do not know to what extent Rouanet was deceived by it. What is certain is that he took it very poorly. This impropriety or more this thoughtlessness had wounded him on a sensitive point. Another colleague made the mistake of recounting the episode in a bit of doggerel verse which circulated about town. This was too much! A duel with pistols ensued. Happily, after the first shot, which grazed both of them, the witnesses, serious-minded colleagues, declared honor satisfied. Rouanet wished to begin again.

Further details on the episode of the goose heart and the ensuing duel is provided by Tinker (1073). It was Rouanet's long time rival Dr Charles Chauvin Boisclair Déléry (1815–1880) who composed and circulated the poem entitled *Le médecin et l'oie*.

As examples of other views on the origin of the heart sound held by outstanding contemporaries of Rouanet three may be cited. James Hope in the first edition of his textbook (1831) attributed the second sound to an impact associated with attainment of full diastolic filling of the ventricle. The great François Magendie (1783–1855) believed (1022) that both major sounds are due to impact of the heart against the rib cage, the first sound with ventricular contraction, the second sound with ventricular filling. The evidence he assembled for this (each bit obviously has an alternative explanation which escaped his attention) was as follows: Attenuation of the heart sound when air and fluid surround the heart; seeming correlation between the strength of the heart beat and the intensity of the heart sounds; difficulty of hearing any sounds from the exposed heart except those which to his mind could be ascribed to impact of the heart on the monaural stethoscope. In regard to Rouanet's theory of the heart sounds Magendie stated as follows: "This explanation is physically inadmissible; it supposes the existence of a void in the ventricles and the two great arterial trunks." It is probably true that Rouanet performed his experiments tensing membranes in air. Magendie apparently questioned whether a snap would result when the tensing was performed in liquid such as blood. (Dock (307) has repeated Rouanet's experiments in liquid.)

The controversy over the origin of the first sound continued unabated despite Rouanet's convincing demonstration. Over a century later a leading cardiologist was still writing as follows (155): "It is generally believed that the principal factor in production of the first heart sound is the sudden increase in the tension of the muscle fibres of the ventricle."

In England a considerable amount of investigation on the origin of the heart sounds was undertaken in the late 1820's and the 1830's. Everywhere, as indicated by the titles of the period, the motions and the sounds of the heart were investigated simultaneously. It had early become evident that to understand both heart sounds

ered on the bladder. At the moment when my fingers which had just squeezed the bladder abruptly relaxed to let the liquid descend, a very marked shock would strike my ear; it was audible as often as I repeated compression of the bladder. Its force was in relation to the height of the fluid column. It was very analogous to the second sound of the heart. I say analogous and not very similar; surely *no one would expect to* find a perfect resemblance here, since the conditions are so different. . . . the pulmonary artery . . . has been submitted to the same experiment with the same *results*.

He described the second type of model in the following words:

The apparatus I most often use is composed of two reservoirs communicating with the heart by tubes. The lower reservoir, designed to furnish the liquid, is raised 3 or 4 centimeters above the organ; its tube connects with the left auricle. The second tube, 2 to 2½ meters long, connects the aorta to the higher reservoir, of which the capacity, like that of the first, is 8 to 10 liters. At the apex of the heart is fitted a third very short tube which carries below a caoutchouc bulb. . . . If one opens the tap of the lower reservoir, *the water enters the heart and rises on the side of* the aorta after having filled the rubber bulb and ventricle.

To produce valve action it is only necessary to *imitate systole and diastole by sudden compression* and release of the rubber bulb. The first of these two movements causes the bicuspid *claquement* to be heard, the second the semilunar *claquement*.

From the results of his experiments he made important deductions about pathologic alterations in the heart sounds: the dull first sound in mitral regurgitation, the loud second sound in arterial hypertension, the timing and valve of origin of murmurs, and so on.

FIRST SOUND. Its force is related to the energy of the contractions of the ventricles and to the exact occlusion of the auriculo-ventricular orifices; thus when presented with unusual weakness, we should look into whether ventricular systole lacks vigor and whether the large valves close their orifices incompletely. In the first case the movements of the heart have small excursion and the apex beat is slight; in the second the heart seems to move forcibly but the pulse remains very compressible, and if the ear perceives a murmur, it is heard most distinctly toward the apex of the heart and not toward the place corresponding to the origin of the arteries. Such was undoubtedly the malady of Marguerite Jolival (48th observation in the work of Messieurs Bertin and Bouillaud): "Beats of the heart soft, but of large excursion; pulses scarcely palpable on the two sides. The heart is very voluminous; the mitral valves are ossified." All these phenomena are very well explained by the *return of blood into the cavity of the auricle*.

The first sound, which is normally dull, can become even more so by thickening and by passage to a cartilaginous or osseous state.

SECOND SOUND. Its force depends (1) on the fullness of the arterial vessels which therefore react with more energy on the blood and push it back more violently against the sigmoid valves; (2) On the rapidity of ventricular contractions. It passes in a given time a greater quantity of blood across the widely opened arterial orifice; the valves, finding themselves widely spread, thereby give with the retrocession of the column of blood more excursion than they would if they were only feebly opened. (3) On the complete occlusion of the orifice by the sigmoid valves; if there remains between them a gap which permits blood to reflux toward the heart, the shock is considerably diminished. The first cause is evidenced by easy compressibility of the arteries; the second by the little energy of the heart and the slowness of its contractions measured by the first silence [systole]. The third should cause an abnormal sound to be heard during diastole of the heart which corresponds to the second silence.

In regard to timbre, the second sound displays considerable changes when the sigmoid valves acquire an abnormal consistency; for example, whenever they are cartilaginous, osseous, or simply thickened, *the superior [second] sound, naturally clear, will* acquire a very striking obscurity which permits prediction of one of these alterations.

Let us hasten to add that the shock, in the sense *of what one ordinarily hears and which results from* the collision of two bodies, is not the only cause of the valve sound. Rather numerous experiments have shown me that all membranes in passing suddenly *from flaccidity to tension always produce a sound* which varies according to the circumstances. Its force is in proportion to those which distend the membrane. Its *éclat* increases with the thinness and inextensibility of the tissue which composes it. *The size, thickness,* and extensibility of the membrane render the sound more dull. The body to which it is attached also influences considerably the qualities of the sound by its thickness, softness and elasticity. *In accordance* with their texture and action the auriculo-ventricular valves combine the most favorable conditions for the production of the sound: they are thin, resistant, inextensible; they pass in an instant *from the most* complete flaccidity to sudden and violent distension resulting from the impulse of the blood and the traction of the numerous tendons which pass from their margins *and ventricular face to attach at the apex of several* carneous columns. Consequently, whether we consider in the valve a surface which goes to strike abruptly against another surface, or whether we see in it an eminently *sonorous membrane subjected to a strong*

and instantaneous force we are obliged to agree that it should produce a sound perceptible to the ear

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and murmurs it was necessary to have a clear view of the sequence of cardiac events. The nature and timing of the apex beat must, it was clear, be established. Although the finishing touches had to await the development of the recording kymograph by Carl Ludwig (1816–1895) about 1847 and of recording techniques by Chauveau, Marey and others, important points were established by experiments such as those of James Hope, famous also as a clinical cardiologist (see p. 18). The technique for one of Hope's experiments is described in this manner: on August 21, 1830, "an ass, of which the pulse and impulse were forty eight per minute, was instantaneously deprived of sensation and motion by a sharp blow on the head. The trachea was opened, a large bellows-pipe introduced, and artificial respiration maintained, while at the same time, the left ribs were sawn through near the sternum, and forcibly bent back and broken, so as widely and completely to expose the heart immediately behind the left shoulder; the whole was accomplished in less than five minutes."

The British Association for the Advancement of Science sponsored studies of the basis of cardiovascular sound in several successive years in the 1830's. Investigations were made by Carlisle (1834) (236), Robert Adams,[19] Law, Green and others (1835) (8), C. J. B. Williams (see p. 23), Todd, Clendinning and others (1836), McCartney, *Adams, Kennedy and others (also 1836)*, and Clendinning (1840) (273). Some of the conclusions were undoubtedly false. For example, the committee of 1835 thought the first sound too long to be valvular in origin and concluded that it is due mainly to ejection of blood over the rough ventricular lining although the "bruit musculaire" might contribute. The studies did further establish that the second sound is related to closure of the semilunar valves and that impact against the thoracic cage plays no role in the genesis of the first and second sounds.

The famous *Jean Cruveilhier* (1791–1874), who held the first chair of pathology in the Paris faculty and whose name is memorialized in the Cruveilhier-Baumgarten syndrome, communicated the findings in an infant with ectopia cordis (318). He could hear almost no sound over the ventricle but heard both sounds over the great vessels. This led him to the notion that the first sound is produced by the flapping open of the arterial valves and the second by *closing of these valves*.

A muscular contribution to the first heart sound or even an origin of the first sound solely on this basis was long favored. In his Croonian lecture of 1809 (1683), *William Hyde Wollaston* (1766–1828), physician, physiologist, physicist and chemist, wrote on the sound produced by contracting skeletal muscle, the "muscular murmur" as he called it. He pointed out that by obliterating the external auditory meatus with the thumb and tensing the muscles of the arm one can hear this muscle sound, thereby demonstrating that muscular effort which appears to be unitary is in fact a composite of contractions occurring at a frequency of 20 or 30 per second. Helmholtz thought it unlikely that the first sound arises predominantly in the myocardium since the sound produced by contracting muscle is usually low pitched.

Auguste Chauveau (1827–1917), veterinarian then in Lyons (Fig. 20), using horses did experiments relating the heart sounds to valve function (253). Chauveau was in the department of anatomy of the veterinary school. Operating on a meager budget he felt compelled to make fullest possible use of the decrepit horses available to him for dissection. He would perform physiologic experiments in the morning before sacrificing the horse for dissection in the afternoon. In one famous series of experiments performed about 1855 he rendered the horse immobile by transfixing the cerebrospinal axis at the level of the medulla. Respiration was maintained by insufflating the trachea with a large bellows. It was a matter of popular interest that the animal in this preparation, although incapable of voluntary motion, could eat sugar and evidence

[19] Robert Adams (1791–1875), surgeon to Queen Victoria and Regius Professor of Surgery at Dublin, was an unusual surgeon. He wrote in 1827 about the condition to which his name, along with Stokes', is attached. He made important studies of mitral stenosis, recognizing the thrill, the high incidence of arrhythmia, and the pulse deficit. He reminds one of the contributions to the understanding of mitral stenosis made by surgeons in recent years.

appreciation for same. Chauveau would open the thorax and introduce the hand into the left atrium via the auricular appendage. By this method he convinced himself that the first sound proceeded from tensing of the atrioventricular valve. In many of these experiments Chauveau had the collaboration of Jean Faivre, a young physician who has the further distinction of having made the first direct intra-arterial measurements of arterial pressure in man (1856). He performed these studies in patients undergoing amputation of a limb and used Poiseuille's hemodynamometer, a mercury filled U tube.

With the help of Marey (see p. 47) Chauveau did the first cardiac catheterization in horses about 1860 using balloon catheters (Fig. 18) introduced through the jugular vein. The time of occurrence of the heart sound was indicated manually and correlation made between intracardiac events and the heart sound (Fig. 19). Visitors to the laboratory expressed amazement that the animal munched hay peacefully as the cardiac phenomena were being recorded (Fig. 20).

In 1860 *Donders* in Holland recorded the apex beat in man and marked the timing of the heart sound by manual means. Thus he did in man what Chauveau had done in the horse. An important result of the experiments of Chauveau and Donders was the demonstration of the nature of the apex beat. Prior to that time it had been held by many that the apex beat is related to atrial systole. Accurate identification was of considerable importance to the accurate timing of murmurs.

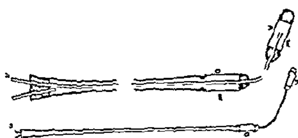


Fig. 18. The prototype of the modern cardiac catheter (1038). The atrial and ventricular balloon are indicated by o and v respectively. One lumen of this double lumen catheter communicates with the atrial balloon and one with the ventricular balloon.

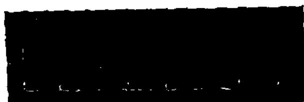


Fig. 19. A recording of intraventricular pressure (*above*) with the time of the heart sound marked manually (*below*) (1038).



Fig. 20. This medallion was subscribed for by a group of Chauveau's friends including Burdon Sanderson of Oxford and presented on January 23, 1903. The medal was designed by Dr. Paul Richer, physician, sculptor and friend of Chauveau. A brochure describing the presentation ceremony was distributed to the subscribers. Abdita naturae scrutatus mente vigenti (meaning "Scrutinizing the obscurities of nature with a lively mind") is an appropriate motto. It is equally appropriate that his most famous experiment is portrayed.

MURMURS. In Dublin *Dominic John Corrigan* (see p. 17) did important early (1829) experiments producing murmurs by compressing arteries in living animals and by compressing lengths of intestine through which he caused water to flow (290). The conclusions of these studies which were directed toward the elucidation of thrill (frémissement cataire, cat purring) as well as murmurs (bruit de soufflet, bellows sound) were stated in this manner by Corrigan:

1. That *bruit de soufflet* is not owing to spasm.
2. That *frémissement cataire* and *bruit de soufflet* are identical.
3. That they do not depend on any particular vital condition of the heart and arteries, but on a purely physical cause.
4. That that cause is an alteration in the motion of the blood, instead of its equable progressive motion *en masse*, it moving as a current producing impulses

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The famous *Jean Cruveilhier* (1791–1874), who held the first chair of pathology in the Paris faculty and whose name is memorialized in the Cruveilhier-Baumgarten syndrome, communicated the findings in an infant with ectopia cordis (318). He could hear almost no sound over the ventricle but heard both sounds over the great vessels. This led him to the notion that the first sound is produced by the flapping open of the arterial valves and the second by closing of these valves.

A muscular contribution to the first heart sound or even an origin of the first sound solely on this basis was long favored. In his Croonian lecture of 1809 (1583) *William Hyde Wollaston* (1766–1828), physician, physiologist, physicist and chemist, wrote on the sound produced by contracting skeletal muscle, the "muscular murmur" as he called it. He pointed out that by obliterating the external auditory meatus with the thumb and tensing the muscles of the arm one can hear this muscle sound, thereby demonstrating that muscular effort which appears to be unitary is in fact a composite of contractions occurring at a frequency of 20 or 30 per second. Helmholtz thought it unlikely that the first sound arises predominantly in the myocardium since the sound produced by contracting muscle is usually low pitched.

Auguste Chauveau (1827–1917), veterinarian then in Lyons (Fig. 20), using horses did experiments relating the heart sounds to valve function (253). Chauveau was in the department of anatomy of the veterinary school. Operating on a meager budget he felt compelled to make fullest possible use of the decrepit horses available to him for dissection. He would perform physiologic experiments in the morning before sacrificing the horse for dissection in the afternoon. In one famous series of experiments performed about 1855 he rendered the horse immobile by transfixing the cerebrospinal axis at the level of the medulla. Respiration was maintained by insufflating the trachea with a large bellows. It was a matter of popular interest that the animal in this preparation, although incapable of voluntary motion, could eat sugar and evidence

[19] Robert Adams (1791–1875), surgeon to Queen Victoria and Regius Professor of Surgery at Dublin, was an unusual surgeon. He wrote in 1827 about the condition to which his name, along with Stokes', is attached. He made important studies of mitral stenosis, recognizing the thrill, the high incidence of arrhythmia, and the pulse *deficit*. He reminds one of the contributions to the understanding of mitral stenosis made by surgeons in recent years.

appreciation for anie Chauveau would open the thorax and introduce the hand into the left atrium via the auricular appendage By this method he convinced him elf that the first sound proceed from tensing of the atrioventricular valve In many of the e experiment Chauveau had the collaboration of Jean Faivre a young physician who ha the further distinction of having made the first direct intra arterial mea urement of arterial pre ure in man (1330) He performed the e studie in patient undergoing amputation of a limb and used Poiseuille s hemodynamometer a mercury filled U tube

With the help of Marey (see p 47) Chauveau d l the first cardiac catheterization in horse about 1860 u ing balloon catheters (Fig 18) introduced through the jugular vein The time of occurrence of the heart ound was indicated manually and correlation made between intracardiac event and the heart ound (Fig 19) Visitors to the laboratory expre ed amazement that the animal munched hay peacefully as the cardiac phenomena were being recorded (Fig 20)

In 1859 Donders in Holland recorded the apex beat in man and marked the timing of the heart ound by manual mean Thu he did in man what Chauveau had done in the horse An important result of the experiments of Chauveau and Donders was the demonstration of the nature of the apex beat Prior to that time it had been held by many that the apex beat is related to atrial systole Accurate identification was of consider able importance to the accurate timing of murmurs

MURMURS In Dublin Dominic John Corrigan (see p 37) did important early (1829) experiment producing murmurs by compressing arteries in living animal and by compressing lengths of intestine through which he caused water to flow (20a) The conclusions of these studies which were directed toward an elucidation of thrill (frémissement cataire cat s purring) as well as murmurs (bruit de soufflet bellows sound) were tested in this manner by Corrigan

1 That bruit de soufflet is not owing to spasm
2 That frémissement cataire and bruit de soufflet are identical
3 That they do not depend on any particular vital condition of the heart and artery but on a purely physical cause
4 That that cause is an alteration in the motion of the blood instead of its equable progressive motion en masse its moving as a current producing impulses

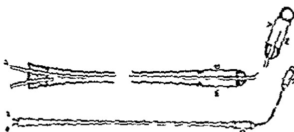


Fig 18 The prototype of the modern cardiac catheter (1863) The atrial and ventricular balloons are indicated by o and v respectively One lumen of this double lumen catheter communicates with the atrial balloon and one with the ventricular balloon

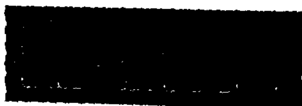


Fig 19 A recording of intraventricular pressure (above) with the time of the heart sound marked manually (below) (1863)



Fig 20 This medallion was subscribed for by a group of Chauveau s friends including Burdon Sanderson of Oxford and presented on January 23 18 The medal was designed by Dr Paul Richer physician sculptor and friend of Chauveau A brochure describing the presentation ceremony was d i d to the subscribers Abdita naturae s nt mente vigenti (meaning Scrutinizing the obscurities of nature with a lively mind) is an appropriate motto It is equally appropriate that his most famous experiment is portrayed

upon the sides of the heart or arteries and according sensations

He added

To produce *bruit de soufflet* it is not necessary, however, that there should be in every instance contraction of some part of the arterial tube, or unnatural dilatation, or when in the heart that there should be narrowing of one of the valvular communications *bruit de soufflet* accompanies simple dilatation of the ventricles From what has been said it will be easily understood that the dilated ventricle and natural sized opening into it bear the same relation to one another as the natural sized ventricle and contracted opening

All of this is indeed remarkable, considering that it was written only ten years after announcement of the stethoscope Corrigan further pointed out that

bruit de soufflet is heard without any lesion either of contraction or dilatation with which to connect it—in hysterics nervous irritable patients in patients suffering from immoderate evacuations from haemorrhage in the very weak and irritable

In the last group Corrigan attributed the murmur to some more vague disturbance of blood flow More precise elucidation had to await description of the role of viscosity of the fluid and velocity of flow in determining the pattern of flow

The second report of the London committee of the British Association (p 42) with *C J B Williams as principal member addressed itself* to the genesis of murmurs Confirmation for Corrigan's observations and conclusions was provided by study of flow through caoutchouc tubes Grating or rasping sounds were best obtained by the action of a strong current on a *knotted* thread across the diameter of the tube Some of the experiments were repeated with water rendered glutinous with size and it was found that the sounds were not so readily produced as with plain water and required a greater force of current This explained the fact that murmurs occur more in the living body in states of anaemia when the blood is thin and more like water

Rouanet (p 39) in his historic thesis on the origin of the heart sounds (1832) discussed murmurs also

Le bruit de soufflet indicates the rubbing ("frottement") of the blood against the orifices of the heart It has stenosis as its usual cause It can also be produced when the ventricle enlarged in capacity or more rapid in its movements causes the blood to pass with much rapidity across an orifice which remains in its natural state This is what occurs in a large number of cases in which the *bruit de soufflet* persists only a short time

Les bruits de scie and *de rape* are only two variations on the same symptom Their cause should be found in a sort of trembling ("tremblotement") and vibration impressed on the column of blood by some body which oscillates with its passage

Can we now recognize which of the four orifices of the heart is the site of origin of the abnormal sound? Two circumstances serve to tell us the time in the heart rhythm when it is heard and the area of the thorax where the ear perceives it most distinctly If the abnormal sound corresponds to the first silence or the contraction of the ventricles its cause is found almost always at the arterial orifices especially if it is a *bruit de râpe* or *de scie* its maximal intensity will be three inches above the cardiac apex and little to the right However this same type of murmur can have its seat at an auriculo ventricular orifice which is poorly closed by the valves it is then toward the cardiac apex that the sound is distinguished with more force What I have just said occurs in the inverse order when the abnormal sound is produced during diastole of the ventricles Most often it is stenosis of the auriculo ventricular orifices which causes it the ear appreciates it best toward the cardiac apex Nevertheless it can happen in certain circumstances that the sigmoid valves altered in texture permit the blood which has just reached the aorta to reflux toward the heart with production of various types of noises of which the maximum intensity is found at the same point of the chest as that of the physiologic second sound

It will be noted that although it did not interfere with his physic Rouanet's understanding of the physics of murmur production was imperfect However after twelve more years spent in the study of cardiovascular sound Rouanet arrived at a more accurate theory of the cause of murmurs He insisted in 1844 (447) that it was in the formation of eddies vortices or turbulence that their basis is to be found The word he used *tourbillons* is noteworthy It is almost superfluous to remark on the facility with which turbulence *(tourbillons)* and murmurs are produced by abnormal dilatation of the arteries stenosis of orifices the presence of blood clots and the insufficiencies



FIG 21 Photograph of a marble bust of Savart by Etax (1841) This is the only likeness of Savart known to be in existence The bust is preserved at the Académie des Sciences in Paris

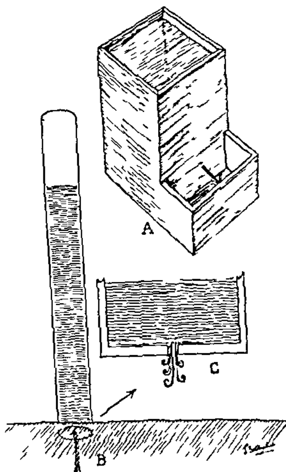


FIG 22 Two of the jet experiments of Felix Savart (author's concept from Savart's description) A and B Over all view of the two models C Detail of orifice plate and vortex shedding in B The approximate dimensions of the model shown in B were as follows he (1852) closed the lower end of a glass tube 6–8 cm in diameter and 2 m long with a metal plate pierced at the middle by a cylindrical opening whose diameter (was) equal to the thickness of the plate

In the 1830's in Paris *Félix Savart* (1791–1841) by diploma a physician but in actuality a physicist and principally an acoustician (Fig 21) did pioneer experiments on sound generation in liquid systems (1852) Figure 22 is an artist's conception of two of the models with which Savart experimented Water is permitted to flow from the reservoir through a small orifice at the bottom (B) A pure tone is generated by this set up through two interlocking forces—the shedding of vortices (or as Savart called them fluid veins) at the orifice and resonance in the fluid column The rate or frequency of vortex shedding is determined by the height of the water in the reservoir The resonating frequency of the water column is likewise a function of its height As the water falls in the reservoir the frequency of vortex shedding falls A sound produced in connection with the vortices at the orifice is intensified to the level of audibility when the natural resonating frequency of the water column matches the frequency of vortex shedding The net effect is that as the level of water falls in the reservoir a series of pure tones of successively lower pitch is produced each tone being separated from the next by a silent period The similarity of the model pictured in Figure 22B to the situation in aortic stenosis and the fact that Savart trained as a physician was in Paris during the exciting period immediately after Laennec, stimulate speculation as to whether Savart's experiments had a medical motivation Aside

from this interesting thought, the importance of his experiments lies in the demonstration that sound can be generated in flowing fluid. During the rest of the last century Savart's 'fluid veins'—call them eddies, or vortices, or even turbulence, if you prefer—are encountered repeatedly in writings on the mechanism of murmurs. In some of his experiments Savart made the "fluid veins" visible by means of a dye, usually indigo, a method exploited by Reynolds 50 years later.

In 1841 Bouillaud wrote: "The precise determination of the conditions under the influence of which the different variations in murmurs are produced constitutes one of the most perplexing problems at present and one of the most difficult in bio-acoustics. It would indeed be desirable if one of our Savarts would apply himself to the solution of a problem which surely is not unworthy of the full attention of the most competent physicists." This would appear to indicate that Bouillaud was unaware of any medical implications of Savart's experiments. However, Savart died in 1841 and his experiments which are perhaps most relevant to cardiovascular sound were reported posthumously in 1854 (1352).

An earlier contribution of Savart has pertinence in connection with cardiovascular sound. In 1826 (1351) Savart studied the velocity of sound in liquids and observed that sound travels much less rapidly in liquid contained in a tube with elastic walls.

In the 1850's *Chauveau*, the veterinarian (p. 42), studied murmur production by essentially the same methods as did Corrigan—he compressed the pulmonary artery in the horse and also studied compressed tubes through which fluid flowed—and came to conclusions identical to Corrigan's. However, familiar with the work of Savart, he attributed the murmur, not to "an alteration in the motion of the blood" (Corrigan) but specifically to "fluid veins"—which amounts to the same.

In 1850 *Franz Alexander Kiwisch Ritter von Rotterau* (1814–1852), versatile professor of obstetrics and gynecology in Würzburg reported (799) studies into the physics of cardiovascular sound and came to the following conclusions as reviewed by Skoda (1395). Kiwisch had found murmurs at the dilatations in caoutchouc tubes. "Water flowing out of a pipe preserves to a certain distance the form of the pipe's outlet, in this way the stream, flowing from the narrow into the wider part of the tube returns to a certain distance the form of the narrow part; now, in consequence of the pressure of the air, the walls of the wider part of the tube have a tendency to adapt themselves to the narrow stream of water, but then elasticity offers a continual resistance to the pressure of the air. Under these conditions, the tube, alternately compressed by the atmosphere and expanded by its own elasticity, is made to vibrate, and by its vibrations a murmur is produced." In the last ten years Rodbard (p. 138) has espoused what is essentially the same mechanism.

Skoda quoted the following conclusions directly from Kiwisch: "'The first sound of the heart is produced by the expansion of the auriculo-ventricular valves, the second sound, by the expansion of the semilunar valves. ... Murmurs arising in consequence of a defect of the valves of the heart are produced in part by the vibration of the rigid valves, but more particularly after the manner described in the experiments with caoutchouc tubes. ... Pressure upon an artery causes a murmur just *beyond* the spot compressed, never at that spot. ... The so-called Nun's murmur—*bruit de diable*—is invariably formed in the carotid artery and not in the cervical veins; it is caused, in fact, by the compression of the carotid by the omohyoideus muscle. Muscular, or any other kind of pressure, will in like manner produce this murmur in other arteries of persons whose blood is impoverished. ...'"

In 1855 (1517) studies of the genesis of murmurs in models were reported by *Theodor Weber* (1829–1899) of Leipzig, later Professor of Medicine at Halle (1228). His father, Ernst Heinrich Weber (1795–1878) of Leipzig was one of the pair of brothers who singly or jointly discovered, among other things, (1) the cardio-inhibitory influence of the vagus nerve (1845), (2) the finite velocity of the arterial pulse wave, disproving Bichat's view that the pulse is felt synchronously in all arteries, (3) the relation of response to stimulation in sensation including hearing (the Weber-Fechner law). In many ways Theodor Weber's study on murmurs was com-



Fig 23 Osborne Reynolds (1842 1912) Reproduction from a painting by Sir John Collier which now hangs in the Department of Engineering of Manchester University (Courtesy of Professor Robert Platt Department of Medicine Manchester)

parable to that of Bondi done about 80 years later (129)

Chauveau collaborator Marey (1830–1904) in his notable *Le circulation du sang* (1881) described (1039) studies in a model simulating coarctation When the feature of the model which corresponded to peripheral arteriolar resistance was low the murmur was less than when it was high A paradoxical increase in flow with increase in peripheral resistance has recently been pointed out by Rodbard (1259) in a similar model In connection with the venous hum Marey pointed out that the murmur was most intense in diastole of the ventricle at which time flow in the large vein in the neck is perhaps most rapid

In the last quarter of the last century *Osborne Reynolds (1842 1912)* Professor of Engineering in Owens College now the University of Manchester in England (Fig 23), made physical experiments of the greatest importance to the understanding of murmur production In two sets of experiments Reynolds demonstrated for the first time with such clarity, and analyzed in fair detail, the phenomena now known as turbulence and cavitation The experiments on turbulence were reported to the Royal Society in 1884 under the title An experimental investigation of the circumstances which determine whether the motion of water shall be direct or sinuous, and of the law of resistance in parallel channels (1263) By direct' Reynolds meant what we would call laminar or streamline flow By sinuous he meant what we would term turbulent flow For these investigations he used the set up demonstrated in Figure 24 and rendered the character of flow visible by injection of a dye into the flowing stream of water The model consisted of a large tank with glass walls filled with water Inside the tank was a long straight glass tube at one end opening into the tank by a

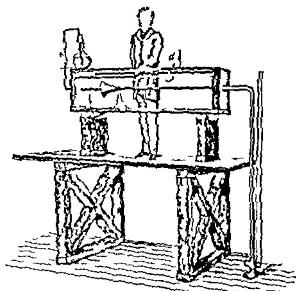


Fig 24 The model built by Reynolds to demonstrate turbulent and streamline flow A long glass pipe with attached funnel end drained off water from the tank when the valve was opened by means of the long handle The wash bottle sitting above the water tank contains dye which—via the small glass tube shown in the sketch—could be injected into the stream of water leaving the tank It is evident how critical velocity could be investigated in such a model From Reynolds publication (1263)

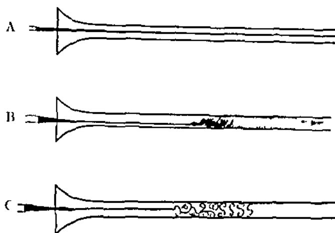


FIG. 25. Patterns of flow demonstrated by the model shown in Figure 24. *A*. Streamline flow at velocities below the critical level. *B*. Turbulent flow at velocities of flow in excess of the critical level. *C*. Semi-schematic illustration of vortices in turbulent flow as viewed by intense light. (From Reynolds' publication (1265).)

funnel and at the other connecting to a drum. A smaller tube permitted injection of a dye into the center of the stream. Using the long handle of the model Reynolds could vary the rate of flow in the tube by opening the valve to a variable extent. He could also vary the diameter of the pipe and the kinematic viscosity (absolute viscosity divided by density) of the liquid.

By this series of experiments Reynolds demonstrated that at low rates of flow the dye maintained streamlines but that above a certain critical velocity flow became turbulent (Fig. 25). Furthermore, the greater the diameter of the pipe and the less the viscosity of the fluid the greater was the tendency to turbulent flow. These considerations Reynolds incorporated in a formula for a value now known as the Reynolds number:

$$RN = \frac{\text{velocity of flow} \times \text{diameter of conduit}}{\text{kinematic viscosity of fluid}}$$

The Reynolds number is dimensionless. If the conditions of the basic model are fulfilled, particularly as to the existence of a long, straight, smooth-walled tube, any liquid will undergo transition from streamline to turbulent flow when the parameters of flow are varied in such a way that the *Reynolds number* exceeds about 2000. The Reynolds formula is rarely applicable in anything approaching a quantitative way in the cardiovascular system. Furthermore, it is not proper to equate turbulence and murmur directly since, among other complexities, an interplay of the disturbed flow pattern and the wall (or protuberance) is important in murmur production. However, the Reynolds formula is a useful catalogue of factors influencing the development of murmurs in the cardiovascular system.

Cavitation was described by Reynolds in 1894 in a communication entitled, "The boiling of water at ordinary temperatures" (1266). He showed that when water is caused to flow with sufficient velocity through a tube with a constriction in it (see Fig. 89) bubbles form at the constriction because of the marked drop in pressure at that point according to Bernoulli's principle. In essence the water boils locally as indicated by Reynolds' title. Reynolds thought the bubbles might be dissolved gases or the vapor phase of the liquid itself or a combination of the two. Beyond the construction the liquid flow moved on in a swirling stream clouded with bubbles. At a point further downstream the bubbles collapse, producing a hissing sound. The structural analogy between the Reynolds cavitation tube and certain lesions of the cardiovascular system, such as aortic stenosis and coarctation of the aorta, suggests a possible role of cavitation in the genesis of the murmurs associated with these lesions. To date this remains only a possibility, however.

A method for approximate quantification of the intensity of the heart sounds and murmurs for clinical purposes was devised by *Hermann Vierordt* (1853-1932), son of Karl von Vierordt (1818-1884), and reported in 1885 (1491). Vierordt had a cylindrical monaural stethoscope, roughly the original Laennec stethoscope, with multiple segments. He would add segments to the stethoscope until the sound being studied was no longer audible. The length of the stethoscope at the point of disappearance of the sound in question was taken as a measure of its intensity.

Vierordt later made important contributions to the understanding of congenital heart disease (1490). Still later he wrote a book on the illnesses of famous people and the role of such illness in shaping history (1493). His textbook of percussion

and auscultation first published in 1884, had gone through 22 editions by 1920 (1492).

THE VOCABULARY OF CARDIOVASCULAR SOUND

Stethoscope was a term suggested by Laennec. It is derived from Greek words meaning "chest" and "to look at."

Auscultation is a word much older than stethoscope. In comparing the sound of the heart to the sound of a horse swallowing, Harvey wrote "... qui sonitum facit et pulsum quendam et auscultantibus et tangentibus exhibet" (see p. 3 for translation). Brown (187) says that Foes was the one who applied the term auscultation to Hippocrates' famous method of examining for hydropneumothorax. Buisson (1776–1805?) of Paris wrote in 1802: "*There are two kinds of audition: one which is passive and involuntary and continually practiced in waking hours ... and a second which is active and produced by the influence of the will on the ear. The term audition is itself appropriate for the first type. I shall designate the second by that of auscultation.*

I define auscultation then as the exercise of the will in audition."

As pointed out earlier (p. 8) Laennec referred to the first and second heart sounds as *bruit ventriculaire* and *bruit auriculaire* respectively. They were also referred to at times as the systolic and diastolic sounds, or the inferior and superior sounds. By 1850 or earlier the current usage became well established.

It is difficult to trace the origin of the designation *aortic, pulmonic, tricuspid* and *mitral* for the major areas of auscultation (p. 72).

The French use of the word *bruit* for the heart sounds is appropriate since they are in fact noises. The German equivalent *Herzton*, sometimes literally translated into English *heart tones* and into French as *tons du coeur*, is inappropriate because of the accepted acoustical practice of reserving the term *tone* for vibrations of pure frequency, i.e., at one frequency or with a small number of harmonics in integer series. The French use the term *bruit* also in the generic sense to indicate both murmurs and heart sounds. Note for example the title of Calo's monograph *Les bruits du coeur et des vaisseaux*. The generic designation used here *cardiovascular sound* has the same significance.

In general *regurgitation*, *insufficiency* and *incompetence* have been used interchangeably. The latter two are less precise than the first. In describing the aortic valve Vesalius in 1549 wrote "Nam obstaculum ne quid penitus regurgitaret effingere fuit impossibile."* Galen, as quoted by Harvey, had used the term *reflux* and Harvey followed suit. Cowper (313) in describing a diseased aortic valve in 1705 used the verb *regurgitate*. Allan Burns used it in his textbook of cardiology (1809).

Incompetence is used particularly by British writers, *insufficiency* by American authors. Some authors tend to use *incompetence* for *regurgitation at an arterial valve*, *insufficiency* for *regurgitation at an atrioventricular valve*. Another quirk is to reserve *incompetence* for what some authors refer to as *relative insufficiency* and to use *insufficiency* for regurgitation due to an organic lesion of a heart valve. The New York Heart Association approves of the word *insufficiency* and *incompetence* and makes a distinction between them. On page 76 of its publication (1179) the following statement is made: "Valvular incompetence is due to dilatation of the valve ring. It is to be distinguished from valvular deformity with insufficiency in that there is no structural alteration in the valve leaflet." The title of a recent article (1357) seems ridiculous at first thought but can be seen to be in the best tradition of the New York Heart Association nomenclature: "Confusion of tricuspid incompetence with mitral insufficiency."

Insufficiency and incompetence are objectionable because they are nonspecific terms; even a stenotic valve is insufficient and incompetent in the performance of *one* valve function, to wit, opening *without* obstruction to forward flow. *Inadequacy* would be as good and as bad a term as *insufficiency* or *incompetence*. In fact Corrigan (293) writing in 1832 stated: "I have been in the habit for some years of describing this disease under the name of *inadequacy of the aortic valves*." He explained that Elliotson in his elegantly written work *Diseases of the Heart*

* For it would be impossible to imagine that the ... of fact would permit *regurgitation* downward.

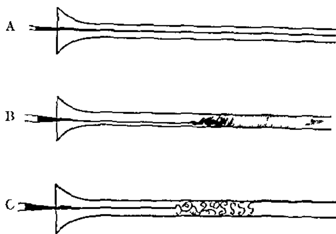


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The French use of the word *bruit* for the heart sounds is appropriate since they are in fact noises. The German equivalent *Herzton*, sometimes literally translated into English *heart tones* and into French as *tons du coeur*, is inappropriate because of the accepted acoustical practice of reserving the term *tone* for vibration of pure frequency, i.e., at one frequency or with a small number of harmonics in integer series. The French use the term *bruit* also in the generic sense to indicate both murmurs and heart sounds. Note, for example, the title of Calo's monograph *Les bruits du coeur et des vaisseaux*. The generic designation used here, *cardiovascular sound*, has the same significance.

In general, *regurgitation*, *insufficiency* and *incompetence* have been used interchangeably. The latter two are less precise than the first. In describing the aortic valve, Vesalius in 1549 wrote: "Nam obstaculum ne quid penitus regurgitaret effingere fuit impossibile."[39] Galen, as quoted by Harvey, had used the term *reflux* and Harvey followed suit. Cowper (313) in describing a diseased aortic valve in 1705 used the verb *regurgitate*. Allan Burns used it in his textbook of cardiology (1809).

Incompetence is used particularly by British writers, *insufficiency* by American authors. Some authors tend to use *incompetence* for regurgitation at an arterial valve, *insufficiency* for regurgitation at an atrioventricular valve. Another quirk is to reserve incompetence for what some authors refer to as relative insufficiency and to use insufficiency for regurgitation due to an organic lesion of a heart valve. The New York Heart Association approves of the word *insufficiency* and *incompetence* and makes a distinction between them. On page 76 of its publication (1179) the following statement is made: "Valvular incompetence is due to dilatation of the valve ring. It is to be distinguished from valvular deformity with insufficiency in that there is no structural alteration in the valve leaflet." The title of a recent article (1357) seems ridiculous at first thought but can be seen to be in the best tradition of the New York Heart Association's nomenclature: "Confusion of tricuspid incompetence with mitral insufficiency."

Insufficiency and incompetence are objectionable because they are nonspecific terms; even a stenotic valve is insufficient and incompetent in the performance of one valve function, to wit, opening without objection to forward flow. Inadequacy would be as good and as bad a term as insufficiency or incompetence. In fact, Corrigan (295) writing in 1832 stated: "I have been in the habit for some years of describing this disease under the name of *inadequacy of the aortic valves*." He explained that Elliotson in his elegantly written work *Diseases of the Heart*

[39] For it would be impossible to imagine that the obstacle would permit regurgitation downward.

suggested the designation *permanent patency of the aortic valve* which Corrigan used in the title of his article In another part of the article he used the term "inefficiency" In 1861 Duroziez (393) spoke of "aortic insufficiency" in describing his double arterial murmur—Duroziez's sign

The best practice, in my opinion, is to use *regurgitation* exclusively, since this more precise term leaves no doubt of the physiologic situation present Admittedly "relative regurgitation" would be awkward, if not absurd "Non cuspal" or "non valvular, regurgitation" might be suitable, or as Wildman (1500) suggests, "secondary regurgitation" Also it might be argued that "insufficiency" and "incompetence" refer to an attribute of the valve, namely, its lack of water tightness whereas "regurgitation" is the functional consequence of the attribute [1] However, since the attribute *per se* is of no significance and only its consequence is physiologically and clinically of note, *regurgitation* remains, I insist, the term of choice

Murmur is derived from the Latin word with the same meaning It is obviously onomatopoetic Occasionally in the past the word *susurrus* borrowed directly from the Latin and meaning a "hissing," was used to mean murmur (e g 1343) *Bruit* and *souffle* are vestiges of the early days when no attempt was made to translate terms borrowed directly from French e g "râle" for "rattle" "bruit de soufflet" for "bellows sound" (blowing murmur) "bruit de scie" for "sawing sound" (a variety of musical murmur), "frémissement cataire" (cat purr) for "thrill" etc

Reduplication, duplication, and *doubling* of a heart sound were used in the past for splitting but are objectionable because true reduplication has not occurred—only components normally present become evident Use of the term might have been justified in an earlier period when systolic clicks and gallops and other sounds in diastole, such as the opening snap and the pericardial constriction snap had not been fully described and distinguished from true splitting

In the last few years the terms *pansystolic* and *holosystolic* have been used interchangeably to refer to a murmur which extends through the entirety of systole Since both prefixes are of

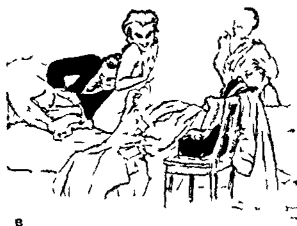
[1] The minor difficulties encountered when one always uses *regurgitation* is illustrated by my statement on p 5 that Douglas found at autopsy aortic regurgitation It was of course an incompetent valve not regurgitation he found

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A



B

FIG 26 *a* and *b* Two cartoons caricaturing use of direct (immediate) auscultation (or at the most the monaural stethoscope) by French physicians *a* Cover of *L'Assiette au Beurre* for March 22 1902 (No 51) Title of issue *Les médecins* Artist Abel Faivre *b* Caricature of direct auscultation "Lange gardien" Artist Hoffmann (Loaned by Dr Lawrence Freeman New Haven)



FIG. ?? c and d. Two more cartoons caricaturing use of direct auscultation and the monaural stethoscope. c, Peinicek. Je pense que l'homme est endormi. From Weber, A.: Tableau de la caricature médicale. Paris, 1936. d, Caricature of physician using monaural stethoscope. Artist's signature not decipherable. (Loaned by Dr. Moses Paul on Baltimore)

Greek origin as is systolic; there is on the basis no objection to either of these terms. However holosystolic is probably preferable since it can mean only what is indicated above, whereas pansystolic also carries the meaning of each. A pansystolic murmur might equally well be one occurring in all systole. Although the latter meaning is usually also true, it is not what the users of one or the other of these terms have in mind.

The term *gallop* dates from Bouillaud who, according to Potain (1224), was using it at least as early as 1847. Evans (442) has suggested the more general use of the designation *triple rhythm* inasmuch as the distinction of gallops from their physiologic counterparts is often difficult. Others (e.g., 942) insist on the desirability of maintaining the term *gallop*.

Some older writers used the term *diminuendo* in lieu of *decrescendo*. They are probably synonymous. The Oxford dictionary so states. Furthermore, the 1911 edition of Grove's *Dictionary of Music and Musicians* comments as follows: "Whether there was originally any difference between decrescendo and diminuendo or not, at present the two terms appear to be convertible."

Battaerd, a student of Einthoven, used the term *cardiophonogram* (61). *Stethocardiogram* has enjoyed some use in the past (1539). In 1910 (??) Lewellys F. Barker (1867–1943) of Baltimore, and in 1911 Kahn (764) used the term *phonocardiogram* which is now preferred and almost universally used. Boerring, Bone and Lockhart (102) and later Dunn (388) favored the term *electrostethograph*.

THE LIGHTER SIDE

As referred to earlier, direct auscultation was practiced by the majority of physicians in France in preference to stethoscopy. The cartoons reproduced in Figure 26 derive from this fact.

Someone has proposed a system of grading of murmurs which from the most subtle and sophisticated at one end of the scale down to the most obvious and blatant at the other goes as follows: Cardiologist's murmur, physician's murmur, surgeon's murmur, administrator's murmur.

In 1848 Dr. Oliver Wendell Holmes (see p. 12) wrote an amusing poem about a young stethoscopist who took his newly acquired tool too seriously.

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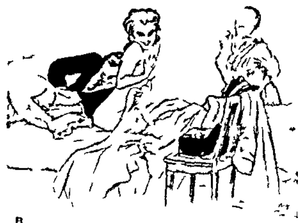


Fig 26 *a* and *b* Two cartoons caricaturing use of direct (immediate) auscultation (or at the most the monaural stethoscope) by French physicians *a* Cover of *L'Assiette au Beurre* for March 22 1902 (No 51) Title of issue *Les médecins* Artist Abel Faivre *b* Caricature of direct auscultation Lange gardien Artist Hoffmann (Loaned by Dr Lawrence Freeman New Haven)

[1] The minor difficulties encountered when one always uses *regurgitation* is illustrated by my statement on p 5 that Douglas found at autopsy aortic regurgitation It was of course an incompetent valve not regurgitation he found

Now use your ears all you that can
But don't forget to mind your eyes
Or you may be cheated like this young man
By a couple of silly abnormal flies

EPILOGUE

In the last century excessive emphasis may have been placed on the findings of auscultation. Preoccupation with cardiovascular sound probably directed attention away from important varieties of heart disease, particularly coronary artery disease. James B. Herrick (1861–1954), who was important in pointing out the clinical features of acute coronary occlusion, recognized the emphasis on physical diagnosis as a factor in the delayed recognition of coronary artery disease (675). Ernst von Leyden (1832–1910), an early student of coronary artery disease, wrote (907) that in his century most physicians seemed *to think that a heart in which auscultation and percussion could reveal nothing abnormal must be healthy and conversely a heart in which one could hear anything unusual must be seriously diseased*. This despite the warnings of physicians such as Latham (p. 19) and of William Stokes of Dublin (see p. 22) who, Herrick states (675), warned against over-estimating physical signs and who said that vital [physiologic] symptoms should be still regarded as of value as they had been in the preauscultatory period.

Mackenzie did a considerable service in emphasizing the importance of the state of the myocardium in determining the clinical course of heart disease. However, he may have underestimated the significance of valvular heart lesions and the auscultatory signs associated therewith. In the English-speaking world cardiovascular sound no longer enjoyed in the first half of this century the intense interest which surrounded it in the last century; phonocardiography never attained quite full respectability as an investigative technique.

Successful surgery of valve lesions has brought a considerable revival of interest in cardiovascular sound. Auscultatory details which previously would have been considered useless minutiae have now become, in some instances, literally matter of life or death. One can examine no better case in point than the so-called "opening snap" of mitral stenosis. Bouillaud, 120 years ago, was probably referring to it, among other conditions, in his *bruit de rappel* and Duroziez in 1861 in the second part of his "four tic-tac-rou." It was beautifully described and assigned its present designation by Rouches seventy years ago. In spite of this, almost no new textbooks of cardiology or of physical diagnosis written in English in this century mention it. The paper of Margolies and Wolferth in 1933 appears to have occasioned little interest and attention. Most textbooks and clinicians persisted in teaching that the double sound heard in mitral stenosis is a split pulmonary second sound. Since 1948, when surgery for mitral stenosis was revived by Charles P. Bailey of Philadelphia, followed by many others, the *opening snap* has in essence been rediscovered and assigned *an important* role in distinguishing predominant stenosis *from* predominant regurgitation and in assessing the grade of mitral obstruction and the results of operation.

Mackenzie maintained that mitral regurgitation *is a benign lesion*. He quoted and heartily subscribed to the statement of Graham Steell that *no one ever dies of mitral incompetence*.' This view held sway in Anglo-Saxon cardiology for the first half of this century. Again the possibility of surgical treatment of mitral stenosis made it clear that mitral regurgitation is a significant lesion if for no other reason than that when combined in any significant proportion with mitral stenosis the results of surgery are less than optimal.

What is likely to be the course of *the study* of cardiovascular sound *in the future*? Will the changing pattern of heart disease and or the introduction of new diagnostic techniques and *refinements of* the old reduce the importance of auscultation? Will the stethoscope be replaced by a more refined instrument or will visual displays of the sound be relied on more to the exclusion of the ear?

Since two major varieties of heart disease—*rheumatic* and syphilitic—can be expected to become *relatively* rare in the rather near future, the usefulness of the stethoscope will decrease greatly. However gallop will continue to be important as a signal of a laboring myo

THE STETHOSCOPE SONG
A PROFESSIONAL BALLAD

by

Oliver Wendell Holmes

There was a young man in Boston town,
He bought him a stethoscope nice and new,
All mounted and finished and polished down,
With an ivory cap and a stopper too.

It happened a spider within did crawl
And spun him a web of ample size,
Wherein there chanced one day to fall
A couple of very imprudent flies.

The first was a bottle fly, big and blue,
The second was smaller, and thin and long;
So there was a concert between the two,
Like an octave flute and a tavern gong.

Now being from Paris but recently,
This fine young man would show his skill;
And so they gave him, his hand to try,
A hospital patient extremely ill.

Some said his liver was short of bile,
And some that his heart was oversize,
While some kept arguing, all the while,
He was crammed with tubercles up to his eyes.

This fine young man then up stepped he,
And all the doctors made a pause;
Said he: The man must die, you see,
By the fifty-seventh of Louis's laws.

But since the case is a desperate one,
To explore his chest it may be well;
For if he should die and it were not done,
You know the autopsy would not tell.

Then out his stethoscope he took,
And on it placed his curious ear;
Mon Dieu! said he, with a knowing look,
Why, here is a sound that's mighty queer!

The bourdonnement is very clear,—
Amphoric buzzing, as I'm alive!
Five doctors took their turn to hear;
Amphoric buzzing, said all the five.

There's empyema beyond a doubt;
We'll plunge a trocar in his side.
The diagnosis was made out,—
They tapped the patient; so he died.

Now such as hate new-fashioned toys
Began to look extremely glum;
They said that rattles were made for boys,
And vowed that his buzzing was all a hum.

There was an old lady had long been sick,
And what was the matter none did know:
Her pulse was slow, though her tongue was quick;
To her this knowing youth must go.

So there the nice old lady sat,
With phials and boxes all in a row;
She asked the young doctor what he was at,
To thump her and tumble her ruffles so.

Now, when the stethoscope came out,
The flies began to buzz and whiz:
Oh, ho! the matter is clear, no doubt;
An aneurism there plainly is.

The bruit de rape and the bruit de scie
And the bruit de diable are all combined;
How happy Bouillaud would be,
If he a case like this could find!

Now, when the neighboring doctors found
A case so rare had been descried,
They every day her ribs did pound
In squads of twenty; so she died.

Then six young damsels, slight and frail,
Received this kind young doctor's cares;
They all were getting slim and pale,
And short of breath in mounting stairs.

They all made rhymes with "sighs" and "skies,"
And loathed their puddings and buttered rolls,
And dieted, much to their friends' surprise,
On pickles and pencils and chalk and coals.

So fast their little hearts did bound,
The frightened insects buzzed the more;
So over all their chests he found
The rale sifflant and the rale sonore.

He shook his head. There's grave disease,—
I greatly fear you all must die;
A slight post-mortem, if you please,
Surviving friends would gratify.

The six young damsels wept aloud,
Which so prevailed on six young men
That each his honest love avowed,
Whereat they all got well again.

This poor young man was all aghast;
The price of stethoscopes came down;
And so he was reduced at last
To practice in a country town.

The doctors being very sore,
A stethoscope they did devise
That had a rammer to clear the bore,
With a knob at the end to kill the flies.

CHAPTER 2

Notes on the History of Respiratory Sound

References to respiratory sound are much more clearly identifiable in the ancient literature than are references to cardiovascular sound. The famous succussion splash of Hippocrates is a case in point. Pleural friction rubs and rales were probably known, and direct auscultation was presumably practiced in their detection. The famous passage in Hippocrates comparing the sound to those accompanying the boiling of vinegar suggests bubbling rales.

An early reference to the Hippocratic succussion (1170) is contained in a letter written in 1679 by Sir Thomas Browne, who described a woman or maid in Suffolk who had a jumbling and fluctuation in her chest, so that when she stood and stroked her chest it might be heard by the standers by, and I once heard it. She dyed, as I remember, about a yeare and half after, and in her chest was found a vessel containing about a quart, as I take it, of a matter like thick whaye, of this Dr. Bur ... now of Woodbridge gave an account to the R.S. [Royal Society] about seven yeares past, and it is printed. (See reference 446.)

Despite its longer history, auscultation has in the case of respiratory sound not fared as well as it has in connection with cardiovascular sound. Whereas much information unobtainable by other clinical methods is provided by auscultation of the heart, x-ray, although it has certainly not replaced pulmonary auscultation completely,[1] has made it less essential. Physical exploration of the chest will remain in the hands of the expert a valuable supplement to the radiologic exploration. A combination of the two can provide a more complete evaluation than either alone.

[1] In a recent case a persistent expiratory wheeze was the only sign of bronchogenic carcinoma. X-rays and all other tests were normal.

The larger share of Laennec's work was in the field of respiratory sound. He described the phenomena of pectoriloquy and egophony and assigned these names to them. In a 28-year-old woman, Laennec found on listening below the right clavicle that her voice seemed to issue directly from the chest and to pass unaltered through the central canal of the instrument. He observed the same phenomenon, which he termed pectoriloquy, in 20 other patients, all of whom had cavities discovered at autopsy. Egophony he described as a high-pitched, sharp, silvery, quavering, jerking sound like the bleat of a goat—and of course the term means "goat sound." He stated: "Its occurrence appears to me to be restricted to subjects suffering from acute and chronic pleurisy with a limited amount of effusion in the pleural cavity."

Rale, meaning "rattle," was used by Laennec in his writing, but in his ward rounds the Latin *rhonchus* was substituted because rale connoted "death rattle" to the French patient of the day. He classified rales into (1) moist rales or crepitation, (2) mucous rales or gurglings, (3) dry sonorous rale or snoring, and (4) dry sibilant rale or whistling. He stated that dry sonorous rales not infrequently resemble the cooing of a dove. This resemblance is often so close that one is tempted to believe that a dove is hidden under the bed. This statement is similar to his comment about the musical venous hum (which he incorrectly supposed to rise in the arteries of the neck) that one might at first think it issued from a musical instrument being played in the apartment below.

Lawrason Brown (1871–1937), physician at the Trudeau Sanatorium, Saranac, New York, has reviewed (187, p. 208 ff.) the history of the use of

cardium in coronary artery disease and in hypertension Congenital malformations will remain with us, and there is reason to expect that cardiovascular sound in these cases contains more diagnostic information than has hitherto been appreciated

Thus, although the stethoscope will become less useful to the cardiologist, it can never be laid aside completely Nor is it likely that it will soon be replaced The human ear, particularly the educated ear, is an instrument to command the respect of all—particularly of phonocardiographers who have attempted to discover in their recordings of heart sounds information which has escaped perception by the ear, or even to display what the ear has been able to detect Improvements on the classical acoustical stethoscope, such as compact electronic stethoscopes using transistors, when used in combination with that excellent instrument the ear, hold much promise for the future

Phonocardiography will continue to occupy a double role (1) of providing permanent, objective, precisely timed records with a certain limited amount of information beyond the ken of the ear, and (2) of instructing the ear in what it should be hearing with the aid of the stethoscope

SECTION II

General and Basic Considerations

cough to elicit râles. The earliest description he could find was by J. Gleize, who wrote in France in 1827. Barth and Roger (see p. 30) wrote an excellent text on auscultation which first appeared in 1841, went through many editions and translations, and had great influence in France in particular. In it, they emphasized the value of what we would call post-tussive râles at the apices in the diagnosis of early pulmonary tuberculosis. Furthermore, they emphasized the value of "expiratory cough," i.e., cough at the end of expiration followed by a full inspiration. Oliver Wendell Holmes in his Boylston Prize Essay (see p. 12) mentions the method, and Bowditch in his *Young Stethoscopist* (see p. 12) emphasized its usefulness. Edward Livingston Trudeau (1848–1915), famed American phthisiologist, probably did more than anyone else to spread use of the method in this country.

Austin Flint (see p. 29) placed great emphasis on the pitch of the breath sounds (466). The term "bronchovesicular" originated with him. He was also originator of a classification of râles which was long used. He divided dry râles into sibilant and sonorous and moist or mucous râles into coarse, fine and subcrepitant (or moderately coarse).

William Wood Gerhard (1809–1872) of Philadelphia, student of Louis in Paris, author of earlier textbooks on examination of the heart and lungs, and important figure in the differentiation of typhus and typhoid, is usually credited with pointing out that the breath sounds at the right apex are louder than at the left (542).

Only scattered notes on the history of respiratory sound are provided here, since it was found simplest to introduce a fuller survey with the discussion of each of the several phenomena of respiratory sound. See pages 475–492 for this discussion.

CHAPTER 3
The Natuı e of Sound

Sound can be defined a con i ting of vibrations which are audible because of proper amplitude and frequency More technically ound in air or liquid at lea t can be defined a den ity waves (or alternate condensation and rarefaction) i e deviations slight to be sure from the re ting' den ity and pressure (ee Fig 27)

If the vibrations of a tuning fork are recorded let us say by mounting a pen on one prong of the tuning fork and running a piece of paper by the pen a regular ine wave i in cribed Since the tuning fork produces a pure mu ical tone of one frequency the ine wave will make a certain number of complete cycles in a second s time For example a tuning fork which produces the note A makes 440 complete cycle in a second and the frequency is said to be 440 c p s (cycles per second)[1] Note that the amplitude of the vibration of the fork the di tance of excursion of the prongs may vary greatly but frequency of the vibration remain the same (within limit)

The frequency at which a tructure vibrates depend on the interrelation hip of mass and re toring force (stiffne or pring if you will) Any structure has a natural frequency of vibra tion dependent on these two properties A large mass on a weak pring will vibrate at a low fre quency in general this is a ituation obtaining in the body A mall mass on a relatively tiff pring will vibrate at a higher frequency in the body arterial wall—di tended under pressure—and bone corre pond more to the latter situation The amplitude varies mainly with the force with which a body is incited to vibrate in its natural period However the amplitude of vibration is not entirely independent of the factors determin ing the natural frequency the greater the mass or the tiffne s the less the amplitude of vibration and vice versa for a given force

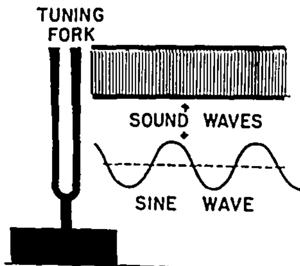


FIG ²⁷ Vibrating tuning fork with chematic repre sentation of alternate areas of conden ation and rarefac tion and corre ponding sinu oidal curve of changing pres ure with reference to resting atmo pheric pres ure (From Ru hmer (1323))

The characteri tics of vibrations are schemati cally represented in Figure 28 These are the fundamental characteri tics whether the vibra tions are those registered in the ballistocardio gram in the low frequency precordial impulse (372 400 401 818 820 1125 1409 1411), in palpable thrills (319 892) the sonic vibrations we are concerned with here or any of many other varieties

There are three main parameters of sound (These are well demon trated in the spectral di play of sound—the sound spectrogram or for heart sound the spectral phonocardiogram) They are (1) *time* (2) *frequency*[2] and (3) *ampli*

[1] Hz is an abbreviation u ed in German and ome other foreign writings U ed ynonymou ly with c p s it is derived from Hertz

[2] Time and frequency are of course interdependent

TABLE 1

Typical intensity levels of familiar sounds

Near Noise Source	Environmental
	db
	140
	2000 μbar
50-hp siren (100')	
	130
Hydraulic press (3')	Boiler shop (maximum level)
Large pneumatic riveter (4')	
	120
	200 μbar
Trumpet auto horn (3')	Engine room of submarine (full speed)
	Jet engine test control room
	110
Chipping hammer (3')	Woodworking shop
	Inside DC 6 airliner
	Weaving room
	100
	20 μbar
Subway train (20')	Inside Chicago subway car
Heavy trucks (20')	
Train whistles (500')	Inside motor bus
	90
10 hp outboard (50')	
	Inside sedan in city traffic
Small trucks *accelerating* (30')	
	80
	2 μbar
Light trucks in city (20')	Office with tabulating machines
Autos (20')	Heavy *traffic (25' to 50')*
	70
Conversational speech (3')	Average traffic (100')
	Accounting office
	Chicago industrial areas
	60
	0.2 μbar
	50
	Private business office
	Light traffic (100')
	Average residence
	40
	0.02 μbar
	Minimum levels for residential areas in Chicago at night
	30
	Broadcasting studio (speech)
	Broadcasting studio (music)
	20
	0.002 μbar

Adopted from Beranek (58)

TABLE 1—*Continued*

Near Noise Source	Environmental
Whisper (5')	Studio for sound pictures
	10
	0
	0.0002 μbar
Threshold of hearing of young ears at 1000 c p s	

oscillogram the features of musical sounds produced by musical instruments—the harmonies are demonstrated in the spectrogram and regularly spaced vibrations in the oscillogram. The presence of harmonic pattern is the basis for the designation of a murmur as musical.

Speech sounds show, particularly in the case of the vowels, harmonic pattern but is not generally considered musical. What physical change occurs in the sound when a vowel is sung rather than spoken? Why is the sound called musical in one case, nonmusical in the other? It is at least in part a matter of vibrato—fine rapid oscillation in frequency through a narrow range—which determines musicality in the *esthetic* sense. This feature probably is missing in the so-called musical murmur; nonetheless they are musical in the more general physical sense.

Except for the relatively uncommon musical murmurs, most cardiovascular sound is not a composite of vibrations of pure frequencies but rather is made up of vibrations more or less randomly distributed over an *appreciable range* of frequency.

The intensity of sound in air away from surfaces follows the inverse square law—intensity *diminishes* as the square of the distance. *If the distance from source to ear is doubled, the intensity is likely to be reduced by 75 per cent.* In bounded media (air or fluid) this law *does not apply*. An air filled tube such as the stethoscope transmits *certain frequencies* with little loss of intensity.

The subject of quantification in sound in general and in *cardiovascular* sound in particular is a complicated one. When *expressed* in terms of sound pressure the unit of sound intensity is *dyne per sq cm*, the *microbar* (One *bar* is one atmosphere, 14.7 *pounds* per square inch, 10^6

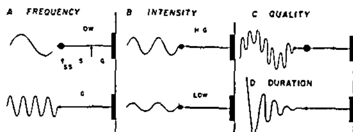


FIG 28 *A* The frequency of vibration is determined by the relation between the mass and the elasticity of the body As shown in the examples schematized here the larger mass vibrates at a lower frequency *B* The amplitude of vibration (intensity) depends on the amount of displacement from the position of rest and therefore on the energy imparted to the system *C* The quality is dependent on the modes of vibration In the example shown two vibrating systems are connected in series The resulting vibration is complex to the extent that there is a major mode of vibration—the fundamental—and a superimposed minor mode of vibration—an overtone or second harmonic *D* The duration of a vibration after the source of energy is cut off is dependent on the level of the energy and the rate at which the energy is dissipated The dissipation is greater if the frictional resistance to motion of the body is greater (Courtesy of Rushmer (1324) and W B Saunders Co)

tude (or intensity) In psychophysical terms the characteristic of duration is obviously related to the parameter of time pitch is related to frequency loudness the magnitude of the auditory impression is related in a complicated that is non linear manner to intensity (see below)

A fourth characteristic of sound is quality, known by the French as *timbre* and by the Germans as *Klangfarbe* (an expressive word meaning "sound color") Quality is a derived characteristic that is it is a resultant of all three of the physical parameters of sound frequency time and intensity In particular, it is a resultant of the relative intensities of the component frequencies of the sound Quality is analogous to color which is also determined by the relative proportions of light of different *wave lengths (or frequencies)* Just as white light can be analyzed into a rainbow spectrum of colors covering the visible range similarly so called white noise can be analyzed into a spectrum of frequencies covering the audible range Just as the relative proportions of components determines whether a color is fire engine red or deep maroon, similarly the relative proportions of frequency components determine the difference in the quality of the note middle C, let us say, as played by a violin and by a trumpet

Musical sounds are characterized by vibrations which are perfectly regular in frequency Usually there are *several frequency components*, each a regular vibration and all at frequencies which bear a simple integer relationship to each other Each component is referred to as a *harmonic* Musical sounds can be resolved into a small number of sine waves at frequencies which are simple multiples of the lowest The pitch of a musical sound is usually determined by the frequency level of the fundamental which is the harmonic component *of lowest frequency and usually of greatest* intensity The quality, however is determined by the relative intensity proportions of this and the *higher harmonics (overtones or partials)* (See Fig 91*B* for demonstrations of harmonics in some musical sounds) Harmonics result from the fact *that vibrating structures, even in the case of* musical instruments generally vibrate not only as a whole but also in separate parts to some extent From Figure 28*C* this will be evident and it will be noted that the amplitude or excursion of vibration tends to be greatest in the case of the major mode of vibration—the fundamental A trained observer can detect by ear not only the fundamental but also one or more overtones in a musical sound (1532)

In the sound spectrogram the several harmonics of musical sounds are represented as horizontal bands at various frequency levels In the oscillogram the regular vibrations are represented as regularly spaced oscillations in the base line with the fundamental dominating as a rule The oscillogram of a musical sound is the composite of several sine waves into which the recording can be resolved by Fourier analysis In the oscillogram overtones with *intensities less than one tenth* that of the fundamental are likely to escape identification, in the sound spectrogram overtones *very much weaker in relation to the fundamental* are identifiable

In the general category of cardiovascular sound there are so called musical murmurs (see Chapter 13) which display in the sound spectrogram and

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parameters since frequency is a *matter of number of* vibrations per unit of time (cycles per second)

mately equal to the mean pressure fluctuation accompanying Brownian motion)

Sound pressure level in db relative to 0.0002 microbar =

$$20 \log_{10} \frac{\text{absolute pressure (dyne/cm}^2\text{)}}{0.0002}$$

Parenthetically it should be noted that one cannot add two values in decibels directly; furthermore a noise of 80 db does *not* have a sound pressure level twice that with a noise at 40 db.

Practically speaking it is useful to keep in mind that, as follows directly from the above formula, *20 decibels represents a 10-fold* difference in sound pressure: 40 db, 100 fold; 60 db, 1000 fold; 80 db, 10,000; 100 db, 100,000; 120 db, 1,000,000. Doubling the sound pressure above the threshold represents a change of 6 db.

In addition, loudness is expressed in terms of the *phon*. However, since loudness has physiologic implications as much or more than physical ones, this system of unitage will be discussed in connection with the properties of the auditory mechanism.

The chart in Table I presents *the intensities of* certain noises familiar in everyday life.

In connection with the intensity of murmurs, size of murmur is a meaningful and useful concept. In evaluating the results of the Hufnagel operation (1077), for example, it was obvious that the *decrescendo diastolic murmur not only was reduced in peak intensity* but also became shorter after operation. A measurement of the area occupied by the frequency-time (SLCG) or intensity-time (PCG) plot seemed theoretically a *better* basis on which to *compare* the murmur before and after operation. It was this measurement which was referred to as *the size of the murmur*. (The sound cardiogram of Rushmer and colleagues (1329) provides a graphic presentation of murmur size directly; see page 84.)

An important measurement is the speed at which sound travels. In air, sound travels at the rate of about 1100 feet (300 meters) per second. However, in water the rate is 4400 feet (about 1100 meters) per second, so that *in the tissues of* the body a rapid rate might be expected. However, this does not prove to be necessarily the case (see Chapter 11). The rate in soft tissues is probably lower and that in bone may be higher.

In music, frequency *or pitch is sometimes expressed in terms of octaves above or below* a given pitch, such as "middle C." In essence this is a logarithmic system. However, this system is of no use in the case of *cardiovascular sound*. *Cycle per second (c.p.s.)* is an adequate—and because it is absolute—a preferred unit. The frequency range of the piano keyboard and its relation to the *range of cardiovascular* sound is *presented in Figure 29.*

Several words in the *acoustical* jargon should be defined briefly. *Transient* is, as the name indicates, a *brief sound*. The so-called heart sounds are transient or, better, in the case of the first and second sounds, at least a combination of transients. *Impact sounds*, again as the *term* indicates, are sounds of collision. Typewriter noise, footsteps, and hand clapping are three examples from day-to-day experience. The valve closure sound may be, *in small part*, impact sounds *produced* by the actual collision of the coapting cusps. *Wide band noise* means sound without harmonic pattern but with *frequency* representation over an appreciable span. Most murmurs are wide band noises.

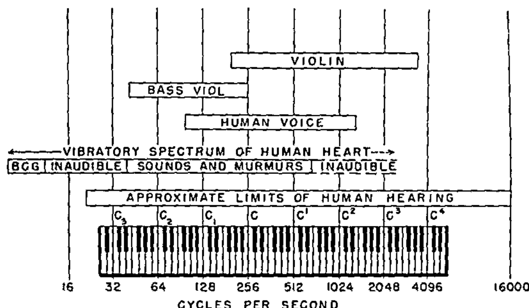


FIG 29 Relation of frequency range of piano keyboard to that of cardiovascular sound the voice and two musical instruments (From Butterworth (204))

dynes/cm²) Referred to is the maximum pressure (above the mean or ambient pressure) which is developed in the process of compression and rarefaction When referred to the threshold of audibility the unit is the *decibel* (db) The sound pressure at the average threshold of human audibility (at 1000 c p s) is taken to be 0 0002 microbar by international agreement This may be considered as zero decibels of sound intensity The decibel scale is logarithmic a feature essential to expressing the wide range in acoustic power which the ear can encompass (in terms of acoustic power a ratio of about 10^6 1 between loudest and faintest)

The decibel is ten times the logarithm to the base 10 of the ratio of two acoustic powers e g if one power is 10 000 times another the difference is 40 db (Originally the decibel scale was worked out on the assumption that the ear obeys the Weber Fechner law strictly and that physiologic loudness varies as a logarithmic function of the physical intensity Approximately one decibel is a just noticeable difference in intensity) The decibel is also used to express the ratio between two sound pressures Since sound pressure is proportional to the square root of the sound power (and the amplitude proportional to the square root of the energy), the sound pressure ratio for a given number of decibels is the square root of the corresponding power ratio In the example given above the pressure ratio corresponding to 40 db is $\sqrt{10\,000}$ or 100, or 20 db [4]

Since the decibel[3] system is a logarithmic one describing ratios not absolute values there is no zero The value expressed in decibels is absolute only with reference to a certain level taken as the base

$$\text{db} = 10 \log_{10} \frac{\text{sound power measured}}{\text{sound power taken as base}}$$

When the unit *decibel* is used by itself in acoustic measurements it is generally understood that it applies to the reference sound *pressure* generally accepted as the threshold of audibility at 1000 c p s i e 0 0002 microbar How small a value this is can be appreciated by realizing that this is a variation superimposed on normal resting atmospheric pressure which is usually about 10^6 bars The threshold level of pressure is approxi

[3] In the mind of most laymen the *decibel* means only a unit of measurement of the intensity of noise Actually the term was carried over into acoustics from electrical communication engineering where it was used originally and is still used principally to indicate *power* ratio

[4] The reason this value is 20 rather than 10 is based on the fact that there is a squared relationship between pressure and power i e power is the square of pressure

$$20 \log_{10} \frac{\text{abs pressure}}{0.0002} = 10 \log_{10} \left(\frac{\text{abs pressure}}{0.0002}\right)^2$$

whenever an amplifying stethoscope is used or when recordings of heart sounds are played back by means of a speaker for teaching purposes, for *example*, Fletcher-Munson considerations enter. In the process of playing a recording sufficiently loud that everyone in an auditorium can hear it, the components of lower frequency in sounds

Fig 30 Relation between frequency intensity composition of cardiovascular sound and frequency response characteristics of the human ear, specifically the threshold of audibility (From Butterworth (204))

which are complex in respect to frequency are relatively exaggerated. As a result the sounds impress the ear as unnaturally low pitched and booming—unnatural as compared to the findings with the stethoscope. If the objective is to reproduce the stethoscopic impression, arrangements for pitch control must be provided.

Several psychoacoustic peculiarities are worthy of note in connection with cardiovascular sound. A loud murmur may *mask a heart sound* occurring just after the murmur, even though it is not superimposed on the heart sound. (Strictly speaking what is referred to is perhaps closer to "fatigue" than "masking.") This phenomenon can be demonstrated by playing the tape recording in reverse. A heart sound not heard on normal playing may be heard on playing in reverse or the converse may be true, that is, a sound just preceding the murmur may escape detection on reverse playing (204).

The difference in the threshold of hearing, depending on whether audition is binaural or monaural, has mainly historical pertinence and interest in connection with stethoscopes. As is demonstrated in Figure 32, binaural audition is

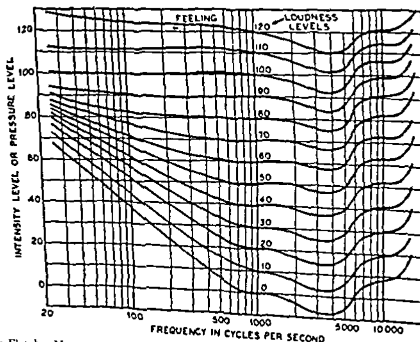


Fig 31 The Fletcher-Munson curves (From Kerr (784)) As the over-all intensity of a white noise is increased the proportionate loudness of the lower frequencies is increased. The frequency response curve for the ear becomes more nearly flat

CHAPTER 4
The Auditory Mechanism

The ear drum is mechanically coupled to the cochlear apparatus by the ossicles of the middle ear. The cochlea is, in essence, a frequency analyzer comparable to the sound spectrograph. The human ear, although in important respects not optimum for analysis of cardiovascular sound, is nonetheless a remarkable instrument. In terms of absolute sound pressure, the average normal threshold of audibility is 0.0002 dynes per sq. cm. This is equivalent to an excursion of vibration in the same range as the diameter of larger molecules and the range of Brownian motion! The sound pressure at the threshold of audibility is, furthermore, about 1/10,000,000,000th of an atmosphere, about equal to the weight of a mosquito's wing. Some persons have sufficiently acute hearing that the noise of molecular collision in Brownian movement is audible (639). The ear drum need move only a distance equal to one tenth the diameter of the hydrogen molecule for sound to be heard; furthermore, the basilar membrane need move only one tenth as far as the tympanic membrane.

All statements about the performance of the ear represent the mean of a very large number of individual testings. For any single individual no smooth curve such as those shown in Figures 30 and 31 will be obtained. Instead, the curve is likely to show peaks of performance at certain frequencies and the threshold may be higher or possibly lower than that indicated and yet be entirely normal.

The average young healthy ear can detect vibration with frequencies between the lower and upper extremes of about 16 and 16,000 c.p.s. However, sensitivity varies greatly through this range. Maximal sensitivity is in the frequency range of 1000 to 2000 c.p.s. Below 1000 cycles sensitivity falls off sharply. To be detected by ear a tone with a frequency of 100 c.p.s. needs to have a sound pressure 100 times (40 db greater than) that of a pure tone at 1000 c.p.s. Most cardiovascular sound is in the frequency range where the ear is relatively insensitive.

The changes in auditory acuity with advancing years concern principally the upper end of the acoustic spectrum. The same is true with many disease processes such as middle-ear disease and otosclerosis. Auscultatory ability is relatively well preserved and may even be enhanced through removal of disturbing or masking noises.

A further complexity of the frequency response characteristics of the ear are represented in the Fletcher-Munson curves. The lines on the chart in Figure 31 are lines of equal loudness. The lowest is the curve for the threshold of audibility and is the same curve as that in Figure 30 except that it is inverted because of different scaling. In making this family of curves, the intensity of sound at 1000 c.p.s., which was interpreted by the ear as having a given loudness, was compared with the intensity which a sound at another frequency must have in order to impress the ear as equally loud. The significant feature of this chart is the fact that, at a high level of absolute intensity, sounds are more likely to be interpreted by the ear as equally loud regardless of frequency content. When the radio is played at low volume, music sounds higher pitched than when played loudly. For best reproduction music lovers must either play their recordings very loudly or preferably (!) have an amplifying and speaker system which favors the lower frequencies.

In clinical auscultation the Fletcher-Munson phenomenon probably is not a factor. However

tant When the first of the two split components is unusually accentuated and reverberating fusion with the second component and reduction in audibility of the splitting result (An example is the lesser audibility of normal inspiratory splitting of the second sound at the base when the aortic component is greatly accentuated in systemic arterial hypertension) It is self-evident that splitting will be less likely be detected when one of the components is of too low intensity or what amounts to the same is of lower frequency composition than is easily appreciated by ear Because of the physiologic properties of the ear a very intense component will render the ear less capable of detecting a second component which would ordinarily be heard and which is separated from the first by an appropriate interval When the second component is accentuated or when it initiated a murmur splitting may be more evident

Loudness level is psycho-acoustic matter is measured in *phons* The phon is derived directly from the Fletcher Munson chart of equal loudness curves The loudness value in phons of a given pure tone is the sound pressure level in db of a pure tone at 1000 c p s which sounds equally loud For example a 200 c p s tone at a sound pressure level of 60 db has a loudness level of 51 phons

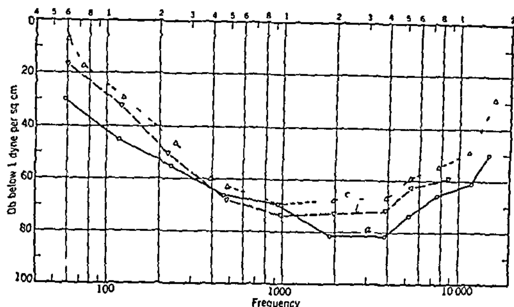


FIG 32 *a* Threshold sensitivity of the human ear for binaural audition in a free field *b* binaural audition in a closed system analogous to the binaural stethoscope *c* monaural audition in a closed system (From Wever and Lawrence (1532))

appreciably more efficient (1532) The data for this chart were collected in a silent room Under ordinary circumstances of ward and examining room the difference would be greater because of masking effects in the case of monaural audition

Experience in phonocardiography brings to attention the fact that the auditory mechanism tends subconsciously to sort out meaningful information from that which is not pertinent from the cardiovascular viewpoint As a result very faint sounds can be *detected by ear* whereas in the oscillogram, since all vibrations are recorded it may be difficult to separate out the vibrations corresponding to a faint murmur In the spectral phonocardiogram it is easier because another *parameter—frequency spectrum—is available*

Other sensory stimuli occurring during auscultation may dull auditory perception During stethoscopy interference from other sensory stimuli should be reduced to a minimum Astute *clinicians in listening for a faint murmur not only* seek as quiet a room as possible—an obvious precaution—but also take a relaxed and comfortable position and sometimes close the eyes (530) In a concert one closes his eyes to appreciate the music The blind are *often good musicians* Jeans (744) claims that pleasure information and protection are more dependent on vision than hearing Furthermore he states that sight developed first in evolution and has an advantage over hearing when the two senses operate simultaneously

The ability of the ear to appreciate "splitting" is of considerable clinical importance How near together can two transients occur and still be interpreted as two? Using a camera shutter set at different speeds one can study this matter The opening and closing clicks of the shutter can be distinguished as two separate sounds at as small an interval as 0 02 sec (752) Actually it is *difficult to quantify minimum perceptible split* since the frequency composition and intensity of the separated components are as important as the interval which separates the components Splitting of valve closure sounds is probably less easily *appreciated than are the sharp clicks of the* camera shutter

The experiments of Helmholtz and of Mayer and Stumpf (744) concerning the greatest number of beats per second that can be heard has relevance *in this connection With pure tones* 41 beats per second (interval of about 0 024 sec) are audible at 96 cycles 58 at 256 cycles (0 017 sec interval) and 107 at 575 cycles (0 009 second interval) Considering that the heart sounds are complex *from the standpoint of frequency* this experience with pure tones is not too disparate from that mentioned above

Training and experience are obviously impor

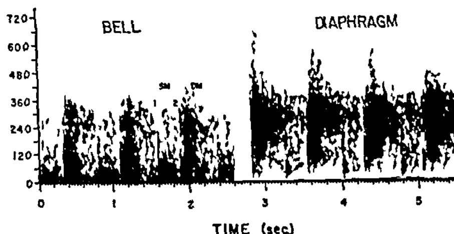


Fig 33 The same murmurs in a patient with aortic regurgitation recorded from a bell and from a diaphragm chest piece with all other conditions of recording and analyzing identical The natural frequency of the diaphragm used in this case was apparently in the vicinity of 300 c p s 1 and 2 refer to the heart sounds and SM and DM to systolic murmur and diastolic murmur respectively

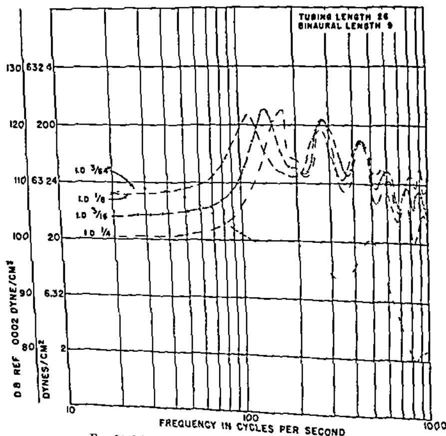


Fig 34 Effect of tubing bore on stethoscope efficiency

Response curves of a stethoscope in which the only variable is the bore of the tubing I D = internal diameter 1/8 inch = optimum caliber of tubing (From Rappaport and Sprague (1943))

CHAPTER 5

The Stethoscope

The acoustically important portions of the stethoscope are (1) the chest piece (2) the tubing which conducts the sound to the ear and (3) the *ear pieces which fit the openings of the external auditory meati*[1]

The bell chest piece applied lightly is optimum for detection of low pitched sounds such as gallops and low pitched murmurs such as that of mitral stenosis The only important consideration in regard to the internal shape of the bell is that *the internal volume should be minimal and the* cross section of the opening should be maximal Both of these features must be compromised to some extent to allow fitting of the bell between the ribs in thin persons and in children and to avoid filling of the bell with soft tissue in obese persons Especially at the lower end of the frequency spectrum *pick up* is better the larger the bell The bell most frequently used is about one *inch in diameter This is probably about as large* as is practical

The reason for applying the bell lightly is that the segment of skin underlying the bell is in essence a diaphragm and the effect of a stiff diaphragm is obtained if pressure is applied Many older clinicians used a bell chest piece exclusively even for the entire cardiovascular examination (Certainly the bell is adequate and in some respects *preferable for the pulmonary* examination) Probably in most instances these clinicians have found the bell adequate merely because they consciously or subconsciously applied pressure when faint high pitched murmurs were suspected

The diaphragm chest piece selectively reinforces components of higher frequency The usual thickness of the diaphragm is about 0 015 in In general the stiffer and the larger the diaphragm the higher is the frequency selected There are limits to how far this can be carried, however since attenuation of the sound, especially at the lower frequency end always occurs also as the diaphragm becomes stiffer (see Figure 33) Some of the advantage of the diaphragm in the detection of faint aortic diastolic murmurs lies in the fact that the diaphragm chest piece has a large opening yet can have a small internal volume because the *diaphragm* keeps out soft tissues Some *(857)* claim superiority for a diaphragm of ordinary thin photographic film This may attain the last mentioned advantages without attenuating the sound as a stiff diaphragm is likely to do

Yet another reason for superiority of the diaphragm in the detection of faint relatively *high pitched aortic diastolic murmurs* is that the intense low frequency components of the heart *sounds and of any other murmur which may* be present in systole for example are attenuated thereby removing a factor of masking or fatigue

The diaphragm or the bell applied with increased pressure is useful in detecting splitting of the heart sounds Whereas at the lower end of the frequency spectrum the transients which make up the sound are intense and tend to run together the separate elements are discernible at higher *levels of frequency*

When in a noisy room one can easily demonstrate to himself the difference in frequency selection by the bell and diaphragm If one shifts rapidly from one to the other by means of the valve of the "quick change" chest piece one notes a higher pitch to room noise in the case of *the diaphragm*

The tubing of the stethoscope should not be more than about 10 inches long and the total

[1] See References 403 753 and 1007

CHAPTER 6
The Art of Cardiac Auscultation

Within this space [the chest] we cannot see. But at this space we can listen and feel and knock and so put it to question whether all be right beneath.

Peter Mere Latham 1847 (843)

Auscultation requires continual practice to maintain fineness of the ear and exact perception of the heart sounds just as the musician needs to practice agility and suppleness of the fingers.

Duroziez 1891 (663)

. . . that most important part of the total system—that between the earpieces of the stethoscope.

Warren 1956 (1511)

Already commented on are the necessity of (1) avoiding interfering sensory stimuli, auditory and otherwise, during auscultation; (2) applying the bell with light pressure for the detection of low pitched sounds. Sometimes when a very loud murmur is present removing the bell only very slightly from the chest or loosening the ear pieces so that there is a leak will attenuate the murmur and permit one to hear the heart sounds proper which were previously drowned by the murmur (618, p. 284). Essentially what is done by this maneuver is to introduce acoustical filtration. The lower end of the frequency scale tends to leak off to a greater extent when a slight opening is present.

It is important to avoid chilling the *patient*, especially in recording heart sounds but also to some extent in auscultation. Otherwise muscle noise of shivering may interfere seriously (*Fig. 40*).

Ambient noise—in the ward or examining room—must be kept to a minimum for best auscultation (609). The examiner should be in a relaxed and comfortable position. He may want to close his eyes in order to concentrate best on the heart sounds (see p. 66) just as one closes his eyes in a concert in order better to appreciate the music. Obviously the patient should remove all clothing from the upper part of the body and facilities for auscultation in both the recumbent and the upright posture should be available. If one merely has the patient push up his undershirt one may encounter a perplexing murmur over the upper chest caused by compression of vessels at the thoracic outlet (182).

As in doing the rest of the physical examination it is a useful convention to work always from the patient's right side. A certain routine in the performance of cardiac auscultation is psychologically conducive to best performance. It is equally important to use always the same stethoscope. This was in the past more important than it is now when stethoscopes are mass produced with a high degree of identity one to another—at least among stethoscopes of the same manufacturer.

In Figure 35 is presented an example *of how one* should *not* go about cardiac auscultation: noisy room, uncomfortable position *of the* physician, patient partially clothed, auscultation from the left side of the patient. Too many persons trying to listen simultaneously is another bad practice. Figure 36 illustrates the correct practice of auscultation.

Various methods must be used to keep infants and children quiet during auscultation or recording. *One writer has* stated that "at times more information is forthcoming from slipping a lollipop into the mouth than a cardiac catheter into the vein" (483). A *child* will *often* permit a careful examination when sitting on the mother's lap even though he was completely unmanageable when placed on an examining table.

Available for pediatric uses are stethoscopes

length of the stethoscope no more than about 18 inches. One must compromise convenience of use (which dictates reasonably long tubing) with the increased intensity of sounds obtainable with shorter tubing.

Tubing with an internal diameter of 1/8 inch appears to be optimal (1243), (see Figure 34). The efficiency is superior to that of 3/16 inch tubing, for example, which has in the past been commonly used. The improved efficiency is particularly evident in the range below 115 c.p.s. Presumably the improved efficiency of the smaller tubing is the result of a smaller volume of enclosed air.

There is a high frequency cut off in stethoscope tubing (see Fig. 34) which, however, is no impediment in most auscultation. Similarly, resonant peaks introduced by the influence of standing waves in the tubes probably do not introduce significant distortion.

Black tubing is professionally and esthetically most desirable.

Snug fit of the ear piece to the external auditory meatus is essential (1430). Fitting of the ear pieces should be made at the time of purchase of the stethoscope. The angle of the metal tubing of the head piece should be such that the tubes are properly directed into the ears. Since the external auditory meati are directed anteriorly, the tubing is angulated accordingly.

Practically speaking, the type of stethoscope which incorporates both bell and diaphragm in one composite chest piece and permits quick change from one to the other by means of a valve is most useful. The chest piece is attached to the tubing in such a way that when the ear pieces are in place in the ears the diaphragm of the dependent stethoscope faces the front of the stethoscopist.

In some chest pieces the rim that holds the diaphragm in place tends to lift the diaphragm slightly off the skin. Preferable is the chest piece in which the diaphragm is shaped in such a manner that this is avoided.

Coupling is an important consideration in connection with sound, particularly in connection with stethoscopes and with the recording of the heart sounds. Even though the energy level of cardiovascular sound may be relatively high, it is not heard externally with the naked ear because of losses in the transfer from the solid to the air medium. The solid stethoscope by coupling directly between ear and chest permitted conduction of sound to the ear for bone conduction. Even here there is, for air conduction, a coupling problem at the end of the stethoscope to which the ear is applied. In the case of the hollow stethoscope, including that with flexible tubing now in use, the relatively limited volume of air contained in the chest piece, tubing and ear pieces is driven by the segment of skin which underlies the chest piece and functions in a manner comparable to that of the driver of a speaker.

The binaural stethoscope is advantageous as compared to the monaural because (1) the ear pieces can provide a more complete seal, (2) binaural audition is more efficient than monaural. In the range of 60–700 c.p.s. binaural audition is about 20 db better—a 10 fold increase in sound pressure.

No stethoscope amplifies the sound. Any advantage which use of the stethoscope has over direct auscultation resides in greater practicability plus the fact that better seal between chest and ear, binaural audition, and a larger effective chest piece are possible.

manubrium and the corpu   terni The second interspace i   of course below the second rib

A will be separately indicated in the appro priate places when valvular heart di ease is u pected *auscultation* should include *in addition* to the four standard area   the following loci (1) *the base of the neck* on the right (re aortic teno is) (2) Erb   area (third left interspace at the ternal border al o known as the   secondary aortic area)   itting and leaning forward (Fig 36*B*) in full expiration (re   aortic regurgitation) (3) the apex *in the left lateral decubitu po ition* (Fig 36*C*) immediately after exercise (re mitral *steno i*) Harvey and hi colleagues (622) rightly emphasize the importance of li tening down the right *ternal border in ca es of u pected aortic* regurgitation

For ome rea on too prolonged au cultation in *one area not only* is profitles but may be confusing as well It is better to li ten several times succes ively in several areas than to inve t the same amount of time li tening once in each area for a longer period

Quiet re piration can be permitted during au cultation and during recording of heart ounds as well It may *of course* be neces ary to have the patient u pend re piration for a time especially *if there i a que tion* of a faint murmur The

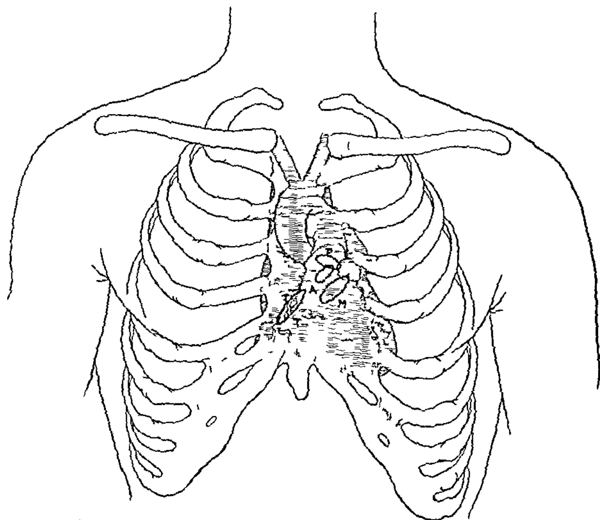


Fig 3 The location of the heart valves with reference to the rib cage Compare with the location as identified in roentgenograms and by calcification in the valve (Figs 60 to 61)



Fig 35 How not to perform cardiac auscultation Noisy crowded room uncomfortable position of examiner working from left side patient partially clad (Inspired by Harvey and Segal (636).)



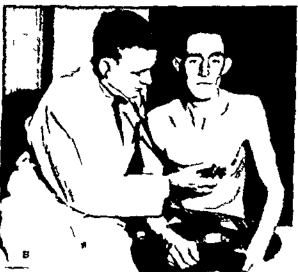




Fig 36 How to perform cardiac auscultation A From right side of patient who is undressed stethoscope in comfortable position *B* Listening for an aortic diastolic murmur in Erb's area *C* Listening for a *mitral diastolic rumble at the apex*

precisely like those recommended for adults but *with* a combination bell diaphragm chest piece of smaller size If the bell is still too large for precise auscultation in infants one can unscrew the Bakelite chest piece and listen using the metal base as a bell

The four cardinal areas of cardiac auscultation and of phonocardiographic recording are as follows (Fig 38)

Aortic area—second right intercostal space at the sternal margin

Pulmonic (or pulmonary) *area*—second left intercostal space at the sternal margin

LLSB (left lower sternal border loosely tricuspid area)—fourth left intercostal space at the *sternal margin*

Apex (apical) *area*—at the apex beat or lacking an apex beat in the fifth intercostal space on the *midclavicular line*

The tricuspid area is variously defined by different writers The apex area is most variable because of the lack of clear landmarks but the definition provided has proved satisfactory in practice

Usually there is no difficulty in identifying the second interspace if it is recalled that the second rib is usually the highest one which is palpable with ease and that it joins the sternum at the angle of Louis i e at the junction between the

hear an *extra sound*. Usually it is best to listen for the over-all canter rhythm which gives the gallop its name.

In timing a murmur, whether systolic or diastolic, it is useful to place the thumb lightly on the carotid pulse. There is too much delay of the radial pulse behind events in the heart to make it useful, and with a very rapid heart rate even palpation of the carotid pulse may be inconclusive. When the apex beat is forceful it can be used for timing purposes. It is rare indeed (p. 413) that a paradoxical motion of the apex beat, so-called diastolic heart beat (1587), will cause confusion.

Approximate clinical quantitation of the intensity of murmurs at the bedside was proposed by Freeman and Levine (481) in 1933 using a system of grading from one to six. Grade I referred to the faintest murmur detectable; grade VI to a murmur loud enough to be audible with the naked ear located at least a short distance from the chest or with the chest piece of the stethoscope removed at least a short distance from the chest. In more detail (888) the grading is as follows: Grade I is a murmur which is likely not to be heard on first applying the stethoscope but becomes evident on close listening. Grade II is the faintest just audible on first applying the stethoscope. Grade VI is audible with the stethoscope removed from the chest. Grade V is the loudest murmur not audible with the stethoscope removed from the chest.

Confusion has arisen from the fact that some use a system of grading in which grades I and IV refer to the faintest and most intense, respectively. Leatham (862) suggests making a fraction of the grade in order to avoid misunderstanding as to whether a base of IV or VI or some other number of grades is used. Thus, I/VI, stated "grade I in VI," would indicate the faintest murmur in the Levine scale.

Lepeschkin (878) has attempted to put grading on a (mu) objective (yet clinical) basis by the design of a stethoscope in which the lumen of the tube from the bell is progressively obliterated. Calibration of the size of the lumen against the subjective impression of intensity permitted construction of a dial from which intensity could thereafter be read. Among several difficulties with this arrangement, most important was the fact that with more intense murmurs conduction through the wall of the bell and valve occurred so that the murmur could not be obliterated even by complete closure of the valve. Another difficulty is that an adjustable valve such as that used, is a high pass acoustical filter; the frequency composition of the sound as well as its over-all intensity is altered. Use of an electronic stethoscope with control of the gain by means of the amplifier may represent a bedside method for quantitating intensity (879, 1108, 1349, 1752). It is doubtful, however, that the end will justify the difficulties associated with the means.

For over a *century* authors have used various systems for graphic representation of auscultatory findings and have recommended various systems for recording *the findings* of stethoscopy for future reference or the *information* of others. Sprague (1429) reproduced a drawing of the sounds and murmurs of mitral stenosis as made in a *patient's* chart by Richard Cabot (p. 16). Segall (1373) has also addressed himself to this matter. In 1957 Brucker (178) published a small monograph in which a system with many excellent features is described. In recording the findings of auscultation, a crude diagram can be worth a thousand words. The oscillographic phonocardiogram has never been able to fill the role. Potentially the spectral phonocardiogram might.

In learning auscultation there is *much to be* said for learning to associate particular sounds with particular lesions without at first analyzing minutely the components of the sound. Sir Thomas Lewis (901) wrote very emphatically on this subject, saying that recognition of the murmur of mitral stenosis should be a matter of learning to know it as one learns to know a dog's bark. Lewis felt strongly that instruction which urged concentration on the timing of the murmur as the first consideration resulted in incompetent stethoscopists. The instructor at first need only say, "This is aortic regurgitation"; analysis of what makes it aortic regurgitation can come later.

Samuel Jones Gee (1839–1911) of St. Bartholomew's Hospital, early student of celiac disease and author of a popular textbook *Percussion and Auscultation* which first appeared in 1870, wrote as follows: "Murmurs were once characterized

performance of a modified Valsalva or Mueller experiment with breath holding may occasion difficulties. Patients, particularly if dyspnea conscious, are likely to make a great effort of "holding their breath." Since what one wishes is merely for them to suspend respiration, it is usually best to say, "stop breathing," not "hold your breath." With the latter request the subject is likely to inhale and "strain down."

Satisfactory auscultation or phonocardiography may be difficult in very dyspneic patients. The difficulties can be obviated in many such patients by having the patient hyperventilate so that he is able to suspend respiration for a time.

The influence of respiration on the heart sounds, specifically the development of splitting in some phases of respiration, is carefully listened for. As will be noted later, the left sternal border, particularly the pulmonary area, is the best location for detecting splitting of the second sound. Splitting occurs normally near the end of inspiration. Occurrence of splitting at another point in the respiratory cycle or persistent splitting, or splitting at the usual time in the respiratory cycle but of exaggerated degree is interpreted as an abnormality.

One should learn to concentrate on the successive time periods of the cardiac cycle separately: the first sound, systole, the second sound, diastole. With experience this becomes automatic. For detecting gallops, however, it is preferable not to concentrate too much on one element or try to

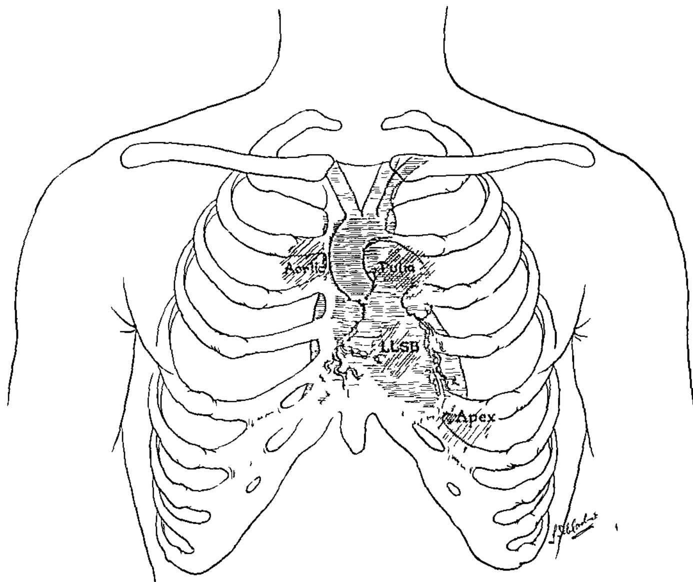


Fig 38 The cardinal areas of auscultation (Contrast with Figure 37.)

CHAPTER 7

Phonocardiography[1]

As outlined in the historical section, all oscillographic phonocardiography is now done by means of electronic amplification and either galvanometer(s) or cathode ray oscilloscope with photographic camera. Spectral phonocardiography is methodologically different as far as the making of the display is concerned and particularly in the introduction of a step of analysis between the recording and the display. However, common to all contemporary phonocardiography are problems associated with (1) the choice of microphone, (2) the pick up of respiration, pulses and other correlative and reference phenomena, (3) amplification, (4) artifact.

MICROPHONES. The two types of microphones now mainly in use are (1) piezoelectric crystal microphone (383, 384) and (2) condenser (or capacitance) microphone. (The electromagnetic microphone has also gained favor with some workers and other principles such as that of variable reluctance [Waters] have been used. At present Mannheimer [1033] uses an electrodynamic microphone.) The advantages of the crystal microphone include inexpensiveness and relatively high sensitivity. Crystals of Rochelle salts were used exclusively in the past but barium titanate has come into use more recently. The advantages of the condenser microphone are (1) stability in respect to temperature changes, moisture and jarring, (2) easy calibration, (3) sterilizability (e.g., in a hot air oven at 240 for 30 minutes). A disadvantage of the condenser microphone for recording in the operating room when explosive anesthetic agents may be in use is the presence of a relatively high voltage between the condenser plates.

Theoretically a contact microphone has certain advantages but other clear disadvantages. The mismatch between the stiff microphone usually used and soft skin and tissue is likely to result in prohibitive loading of the skin with bothersome distortion. The microphone of Groom and his colleagues (607) is designed on the capacitance principle, the skin being used as one plate of the condenser. One can even record from the surface of the heart without touching it, thereby avoiding loading[2] of the surface from which the recording is made. As a research tool it may become the standard of reference since it should provide valuable information on the precise character of the vibrations at the level of the skin. There is no air conduction of the sound, no moving parts, no loading of the skin. Room noise has less effect. The microphone is exceedingly sensitive. On the other hand, hair and sweat will short out the microphone and calibration is difficult or impossible.

Fortunately the air seal of the microphone, such as the condenser type, with the surface of the chest results in as much as 20 db attenuation of ambient noise. Special soundproofing is usually unnecessary for phonocardiography. However, a recording area remote from elevators, the clatter in kitchen, traffic in corridors and conversation of waiting rooms is essential.

Manual application of the microphone is usually superior to the use of a strap (1033). Too forceful application of the microphone is not only uncomfortable to the patient but also attenuates the sounds with alteration of frequency compo-

[1] See the Technical Appendix, p. 499 ff. for more detail. See the Historical Section, p. 30 ff. for background information. Also see References 531, 601, 606, 530, 937, 1031, 1166, 1515.

[2] This difficulty is avoided by covering the skin with light foil.

according to their acoustic qualities, whether blowing, filing, rasping, sawing, but these are vain distinctions, in order to render murmurs serviceable in the diagnosis of disease we now regard two only of their properties namely, their Place and Time" I cannot agree that distinctions in quality are as vain as Gee claimed Many murmurs which are identical as to distribution over the surface of the chest and in the cardiac cycle are quite different in quality, and the difference in quality is a diagnostically helpful feature

Similes are useful in teaching auscultation but care must be taken to choose familiar sounds for the analogy The early designations for murmurs were similes *bruit de soufflle*, bellows sound blowing murmur, *bruit de lime*, *bruit de rape*, *bruit de scie*, file-, rasp-, and saw sounds, varieties of musical murmurs Vivid similes have from the first enriched the literature on clinical auscultation of the heart and are an integral part of the art of stethoscopy A keen ear for fine differences in quality and rhythm in cardiovascular sound is a valuable asset to the cardiologist Some of the most effective teachers of clinical auscultation are those who can reproduce the sounds vocally Similes and onomatopoetic devices are useful in teaching cardiovascular sound The following paragraphs are quoted from a charming essay by Geoffrey Bourne (143) The piece is entertaining and instructive although many of his similes will be unfamiliar to American students

> The diastolic aortic murmur heard best in most cases down the left sternal border and to the apex has the high pitched rushing and persistent quality of water escaping under pressure from a hose over a weir or from a leaky lock gate The diastolic murmur of mitral stenosis—not the presystolic—has the low pitched rumbling quality of a wooden wheelbarrow trundled over cobble stones Either of these murmurs may be loud and easily heard or distant and elusive but the weir may be distant too and the wheelbarrow in the next garden but one The quality of the sound is unchanged in both types of sound whether remote or near bucolic or pathological
>
> A less rural simile is applicable to the forcible harsh, brief systolic murmurs caused by the powerful ventricular expulsion of blood through a rigid and narrowed valve Examples of this are found in aortic stenosis pulmonary stenosis, and coarctation of the aorta The sound here suggests the deliberate forceful and short blasts of steam given off by the Scottish express starting from rest at King's Cross The sound may be heard from the other end of the station or from farther off still, and may vary correspondingly in volume but the character of both steam blast and murmur is very similar When this same type of murmur is due to coarctation of the aorta it is naturally much fainter than in the other two conditions
>
> Another sound—a rarity—heard when an aortic valve cusp is severely ruptured, perforated, or perhaps even everted as a result of syphilis or subacute bacterial endocarditis is a diastolic croon This murmur closely mimics the coo of a nearby and rather hoarse wood pigeon which has been restricted to one rhythmic falling note instead of the usual three or four
>
> The drum beat of a dance band playing a two step is a measurable fraction of time in advance of the shuffle of the dancer's feet upon a well chalked floor The double rub of the pericardial surfaces is similarly appreciably delayed behind the beat of the first and second heart sounds Sometimes the rhythm of the friction instead of being to and fro is triple and to modify the metaphor the dance becomes a waltz not a two step the third sound of the triplet being caused by auricular systole Once more it must be stressed that the quality and timing of the sound are the important points The volume varies The shuffle of the dance may be quite loud or if the ballroom door be shut may be heard only by close attention

In a similar vein Bean (75) writes as follows

> *Bruit de moulin* the noise of the mill is a very happy simile since the combination of water splashing rhythmically and a background sound of wooden machinery and running water is elegantly reproduced in miniature by the sound of pneumopericardium as any knows who has listened to both

It is in connection with musical murmurs and with murmurs audible at a distance from the chest that greatest imagination and resourcefulness has been displayed in the invention of ingenious metaphors In part this is due to the fact that patients and other laymen in hearing these sounds have devised a simile unfettered by existing medical clichés and conventions of phraseology

at 15 cycles has dropped a maximum of 20 db below the response level at and above 30 cycles

One can put the reference data on the same magnetic tape track as the sound using a carrier at a frequency level of let us say 3000 cycles well removed from the range of cardiovascular sound However since this combination of the EKG and respiration signal with the heart sounds impairs the usefulness of the latter recording for teaching purposes it is desirable in many situations to have a tape recorder with at least two channels so that the sound can be recorded separate from although synchronous with the other information Of course by filtration the carrier signal at a higher frequency can be removed in a re-recording step and the sound recording recovered for teaching purposes and other audition

In *oscillographic phonocardiography* the frequency response of the system is of great importance and the matter cannot be entirely disregarded in spectral phonocardiography either Rappaport and Sprague (1244) suggested two types of recordings (Fig 40 A) one they called *stethoscopic*[3] and represents roughly the character of the sound as they are presented to the ear by the stethoscope (there is a sloping response curve with reference to frequency response rising at the rate of about 12 db per octave) the other they called *logarithmic*[3] because components at higher frequencies are accentuated (or the low frequency components are attenuated) in logarithmic fashion to approximate the auditory impression of the sounds (the response curve has a slope of about 18 db per octave) Recordings of the latter general type are sometimes called ear like (Ger Öhrahnliche or Gehorsähnliche) Leatham (859) call this type of recording high frequency (Fig 40 *B*) logarithmic is in his opinion too forbidding a term for physicians There is in addition the so called *linear phonocardiogram* which has minimal low frequency compression Because of the intense low frequency components in the precordial vibrations linear phonocardiograms have very limited usefulness Many e g Johnston (754) and Luisada and Magri (989) have made a particular study of the

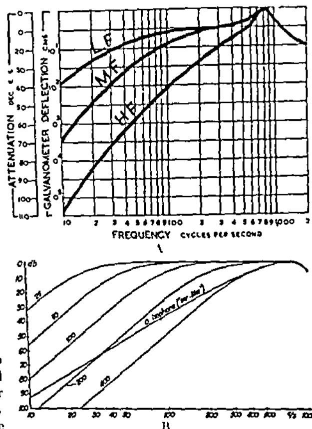


Fig 40 A schematic representation of the frequency response characteristics of several phonocardiographic systems *A* Leatham's high mid and low frequency system (859) *B* Mannheimer multiple filter system (1031) These various systems are necessitated by the fact that large intensity range of cardiovascular sound particularly the very intense low frequency components and the relatively faint higher frequency components cannot be displayed simultaneously in a single oscillogram A second motivation is the ability to obtain approximate information on the frequency composition of heart sounds and murmurs by the examination of oscillographic displays of various frequency bands The system of Maass and Weber (1001) is similar to that of Mannheimer Luisada's selective phonocardiography (993) uses a filter which cuts off those components above as well as below a certain frequency band

low frequency precordial vibrations and Eddleman and colleagues (400 401) refer to the records as kinetocardiogram

The logarithmic (high frequency) phonocardiogram has been useful for displaying murmurs and the stethoscopic (mid frequency) phonocardiogram for the heart sounds proper

Unless the frequency response characteristics

[3] At first these two types of recordings were achieved by the use of two microphones of different frequency characteristics later by electronic filtration

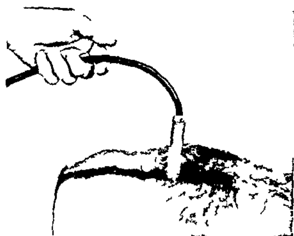


FIG 39 This manner of holding the microphone reduces extraneous noise due to hand tremor and standardizes the pressure of application of the microphone—its weight

tion, in effect, one creates a stiff diaphragm of the skin The pressure with which the microphone is *applied is a variable which should be controlled* A practical way to keep it relatively constant is by holding the microphone as shown in Figure 39 The pressure of application is the weight of the microphone and is therefore constant from patient to patient and from time to time or from area to area in the same patient The method has the additional advantage that slight hand tremor has little effect being *absorbed by the flexible* cable Unfortunately the system breaks down when one records from the axilla since the microphone itself must be held in such a site *Cleaner looking oscillographic* phonocardiograms may be obtained when the microphone is held firmly with a strap or suction cup rather than lightly with the hand This fact may be the result of attenuation of bothersome *low frequency* vibration by a diaphragm effect of the skin underlying the chest piece In the SPCG because of greater dynamic range low frequency attenuation by electronic or mechanical artifices is less essential

The presence of much hair on the chest may be troublesome Crackles may be introduced and it may be difficult to attain an intimate seal of the microphone with the skin In such instances the chest can be wetted to flatten the hair and the microphone held in the usual manner Simpler and usually more effective is to hold the microphone firmly to the chest directly with the hand This procedure both holds down the hairs and ensures good skin seal Vaseline or preparations containing lanolin which are available commercially for dressing the hair have also been used with some success

The lowest possible level of electronic noise in the amplifier is essential Otherwise, in attempting to pick up very faint murmurs and sounds, amplifier noise may predominate A favorable "signal to noise ratio" is necessary What the electronics experts term valve noise is particularly troublesome at the first valve With full gain on the amplifier the "base line" of the oscillographic record should be smooth and the background of the spectral phonocardiogram clean The microphone should of course, be similarly silent

Recording heart sounds *on magnetic tape* has great usefulness for teaching purposes and is a permanent record Phonocardiograms either oscillographic or spectral can then be made as desired and other operations such as selective filtering accomplished The ability to put reference recordings such as electrocardiogram and pneumogram on the tape also by means of frequency modulated carriers has facilitated greatly the accumulation of libraries of recordings which can be variously studied The techniques for analysis and display of the sounds change but the recordings are always available for restudy

In the case of all the spectral phonocardiograms published here the sounds were first recorded on magnetic tape and the display made later The electrocardiogram and respiratory mark will be similarly recorded on tape Any pressure pulse, such as the jugular venous pulse or carotid pulse can similarly be recorded on the tape after translation into an electrical signal by appropriate transducer See Figure 50 for spectrogram made *from tape recordings* which used the jugular and carotid pulses *for time reference*

In this laboratory most recording has been done at a tape speed of 7½ inches per sec occasionally at 15 inches per sec (Butterworth (204) has championed slower tape speeds e g 1⅞ inches per sec) Without imposed "low frequency compression" the response characteristics of the entire system from microphone through to magnetic *tape are flat* above 30 c p s At 20 cycles the response had dropped 5 to 10 db and

may be exceedingly loud. In the same patient there may be an exceedingly intense systolic murmur of aortic stenosis and a barely perceptible diastolic murmur of aortic regurgitation. For the oscillogram to record both in their true intensity proportion, the excursions representing the systolic murmur might have to be several meters high in order for the diastolic murmur to show up with an amplitude of one or two millimeters. In practice, voltage limitation is employed in such instances to place a ceiling on the systolic murmur and bring out the diastolic. The spectral phonocardiogram by spreading out frequency and thereby reducing the intensity to be represented to that present in each frequency zone attacks the problem of intensity display in a different manner and more nearly succeed in representing sounds in their true intensity proportion. Voltage limitation truncates loud murmurs and distorts their intensity time contour as represented in the oscillographic PCG. Again, the SPCG displays more faithfully the true shape of the murmur, a feature of no small usefulness in identifying the cause of the murmur and relating the murmur to hemodynamics.

A problem distinct from calibration, although related to that of standardization in oscillographic phonocardiography. It is a laudable objective to have uniformity of instrumentation such that recordings from different laboratories can be compared. A beginning *on standardization* has been made (81, 804, 1118). *Until certain* standards are generally accepted and *given the* stamp of approval by an *international body*, such as the World Congress of Cardiology, *care must* be taken to define the characteristics *of the instrumentation* used in any serious *phonocardiographic* investigation.

What *reference recordings* are most useful for correlation with the heart sound recordings? The electrocardiogram is necessary in all cases as a basic minimum. Standard limb lead II is usually used. The objective is to obtain sufficiently large complexes for timing purposes. Other leads must be tried when, let us say, the QRS is small in lead II. In special instances, when the P wave must be recorded for correlation with atrial sounds and murmurs, the lead with the largest P wave must be sought.

Orias and Braun Menendez (1166) and many others (1098) have championed the venous pulse (699), specifically the jugular pulse; it was their contention that the timing of diastolic events was most satisfactory by correlation with venous pulse. More recently it has been a fairly general opinion that the difficulties in recording the venous pulse and the lag between the events in the heart and the reflected changes in the neck make it objectionable for routine recording. One study (402) arrived at the conclusion, probably erroneous, that the physiologic third heart sound is a physiologic opening snap of the AV valve. The conclusion was based on temporal correlations of the heart sounds with the jugular venous pulse and the lag may have been responsible for the results. Kuo *et al.* (828) found a delay of as much as 0.14 to 0.20 sec. in transmission of the venous pulse wave to the *jugular vein*.

Another objection to use of the jugular pulse is the technical difficulty of recording, *especially if* cups or tambours are used. Piezo-electric transducers can be applied directly to the overlying skin. Furthermore, Holldack and Wolf (700) have *made* use of a recording device consisting of a light paper arm or "rider" which is *applied to the* skin overlying the *jugular vein and which interrupts* to a variable degree a beam of light passing from a source to a photoelectric cell.[4]

Leatham (849) has emphasized the usefulness of the indirect carotid pulse tracing. Particularly is the arterial trace helpful in the separate identification of the aortic and pulmonary closure sounds—a matter of considerable clinical importance by the evidence of Leatham. One must allow *for a* delay of 0.02–0.04 sec., part of which occurs *in transmission* of the pulse to the neck, part in the recording device. If a cup is used for the pick up of the carotid pulse application *of the cup* is critical (706). If improperly *applied* one may actually obtain a wholly *or partly inverted* pulse curve. An initial dip may leave one uncertain as to the point at which onset of the anacrotic limb should be placed. The contour of the pulse as recorded *from a somewhat* haphazard application of a *cup or tambour type* pick up is not reproducible and cannot be depended upon to give

[4] Apparatus made by Firma Schwarzer in Germany.

of the oscillographic recording system is satisfactory, errors in display may be produced, for example, the presystolic murmur of mitral stenosis may not seem crescendic because the higher frequency components which make a contribution to the total intensity and increase with the crescendo are not properly represented For a similar reason the systolic murmur of aortic stenosis may not be properly diamond shaped At this point a plea is made for all workers publishing results of phonocardiography to *determine and report the frequency response characteristics of the entire system used*

The frequency parameter has been manipulated in other ways Mannheimer and Stordal (1031) by selective filtration split the sounds into five separate although overlapping frequency bands (Fig 40), representing the information in each band as an oscillogram His bands were as follows 0–100 c p s , 50–175 100 250, 175–400 250–500, 500–1000 Contrary to what is generally thought, the filters used by Mannheimer attenuated the signal to a variable degree only at the *lower* end of the frequency spectrum (see Fig 40 C) The output from the five filters was recorded in a parallel manner by a multi channel recorder This method was termed "calibrated phonocardiography", features of intensity calibration are discussed later Maass and Weber (1001A) specified a multi filter multichannel phonocardiographic system comparable to Mannheimer's and Holldack and Wolf (706) make extensive use of a system which similarly splits the sounds into five frequency bands in roughly the same range Luisada (993) has used what is, for practical purposes the same method but which he calls "selective phonocardiography" Luisada does make use of both high and low cut filtration, that is, he attenuates the components of the signal in a frequency range above as well as below a specified range

Ljunggren (959) provided a critical appraisal of the Mannheimer Stordal system which in essence consisted of crystal microphone pre amplifier filter system amplifier recorder He pointed out the risk that distortion in the form of *de novo* generated overtones might be introduced at the pre amplifier stage because of overloading by high amplitude vibrations in the lowest frequency band (0–100 c p s) This distortion would have the result that in the recordings from higher frequency bands components which do not actually exist would appear Too long a time for build up and for decay of filters may introduce time distortion These important considerations must be kept in mind in any system involving filters including spectral phonocardiography

The oscillographic phonocardiogram can be registered by means of galvanometers or the cathode ray oscilloscope In the case of both a photographic record can be produced Direct written records are possible in systems using galvanometers Holldack and Wolf (706) and others (1325) have used direct writing galvanometer recorders with seemingly satisfactory results Direct writers with excellent frequency response characteristics (e g , the jet ink writer of the Elema Co ,) are now available

Intensity calibration in phonocardiography presents many problems (586) there are variables in the sound transmitting properties of the chest among patients and in the same patient under different circumstances (see p 147 *ff*) There may be variability in the performance of the microphone which often is not easy to calibrate in the first place The electronic amplification must be quantitated Complicating matters greatly is the fact that changes in intensity must be considered in relation to frequency Sounds of quite different frequency composition may be of identical overall intensity

For calibrating purposes Luisada and Gamna (985) use a sound generator strapped to the chest in about the left anterior axillary line McGregor Rappaport Sprague and Friedlich (1060) attempt to calibrate only what reaches the surface of the chest and in fact only what came out of the microphone An electrical calibrating signal was introduced proximal to the microphone Sloan and Greer (1398) used a similar method by introducing at the input stage a standard signal at 50 c p s and 25 db above the threshold of audibility

The dynamic range of cardiovascular sound is enormous In the extreme instance the loudest sound is painful to the ear and the faintest is located at the threshold of audibility or below In congenital heart disease in children the murmurs

mended from time to time and are used especially for investigative purposes. Obviously recording is indicated from any area where stethoscopy indicates sound of interest to be located.

Duchosal suggested a system of labelling records using letters and numbers, e.g. 2LS = second left intercostal space 3 cm from the midsternal line; 4M = midsternum at level of fourth intercostal space. Some may find it useful to have printed forms or a rubber stamp, so that by means of a number the precise site of each recording can be indicated during the recording procedure.

Recordings from the esophagus are made either by attaching the transducer to the end of a catheter[*] or by having the subject swallow a small microphone (683). By either method it is desirable to insure contact with the wall of the esophagus by means of inflation of a balloon with water or air. An atrial heart sound is always demonstrable by esophageal recording unless, of course, atrial fibrillation or other dysrhythmia with absent atrial systole is present. The heart sounds as recorded from the esophagus are of lower pitch than those recorded at the surface of the chest. This fact has raised the question of whether the thoracic cage may not be incited to vibrate in its own natural period and thereby contribute higher frequency components to the heart sound. Sach (1337A) attached his microphone to the external end of an esophageal tube 70 cm long and with an internal diameter of 3.5 mm. A lateral opening 15 mm long, covered with a thin rubber membrane, was provided near the internal end. The diastolic murmur and especially the presystolic (Austin Flint) murmur of aortic valve disease was louder in the esophagus although the systolic murmur was louder at the precordium. In one case of presumed mitral regurgitation the murmur was demonstrable only in the esophagus.

Recording directly from the surface of the heart and great vessels (1565) has difficulties because of the almost perpetually moving surface, with possible introduction of vibrations which are not of physiologic significance, and because of loading of the surface by the microphone. Bertrand, Milne and Hornick (96) used suction to hold the microphone on the heart. Especially in man, allowing the microphone to rest on the heart and ride with it may be satisfactory, depending on the area from which the recording is being made and the type of microphone available.

Intracardiac phonocardiography can be said to have had its crude beginnings with the intracardiac pressure recordings of MacLeod and Cohn (1015), who found high frequency vibrations equivalent to the heart sounds superimposed on pressure recordings. Dock (380) took delight in pointing out that the *vibration corresponding to the first sound had maximal amplitude in the vicinity of the AV valve. Recording of the rapid* pressure transients within the heart has been attempted by means of a barium titanate (895) or miniature electromagnetic (1121) transducer on the tip of a catheter, a condenser microphone with one pole at the tip of the catheter and the other the body itself (1396, 1397), or a transducer[†] located on the external end of the catheter and communicating with the interior through the blood filling the catheter (979). If one uses an internal transducer, a double lumen catheter permits combination of intracardiac phonocardiography with cardiac catheterization for purposes of blood sampling and pressure recording. The information obtained by means of a transducer at the external end of a catheter must be interpreted with utmost caution because of distortion, unknown and known. This laboratory has been disappointed in the performance of the available intracardiac transducers of the barium titanate type. Disturbingly sensitive to bending of the catheter, to striking at a distance from the tip and even to minor temperature changes and relatively insensitive to localized pressure changes at the tip, they are suspect as true indicators of high frequency *pressure fluctuations* within the heart. When all factors are considered, the use of an external transducer on a venous or arterial

[*] This method of esophageal pick-up dates at least from 1899 when Hofmann (?) in Germany and Richardson (1772) in England passed a tube into the esophagus and listened at the external end. In 1913 Lian (911) used the same method of esophageal auscultation. It is of note that with the ear he heard an atrial sound in only one of 25 subjects.

[†] [illegible] has used the Statham P23D strain gauge.

dependable diagnostic information, as in cases of aortic stenosis It is true that timing not contour, is the information sought in the use of carotid pulses in phonocardiography A modification (203) of the carotid pulse tracer of Duchosal (379) avoids some of the technical difficulties of the cup type of carotid pick up and provides more meaningful records from the standpoint of contour Around the neck is placed a collar consisting half of elastic "moleskin" and half of a narrow inflatable cuff which is maintained at a pressure of about 20 mm Hg during recording The interior of the cuff communicates with a Statham strain gauge *With this system it is difficult or impossible to record the venous pulse simultaneously*

Respiratory phase should also be indicated in recordings Effects of respiration on the splitting of heart sounds and on the intensity of certain murmurs is of great diagnostic significance For recording respiration, belts around the chest with mechanical or strain gauge pick up and face masks with various pick up devices such as a hot wire variable resistance have been used Holldack and Wolf (706) use the same method as for venous pulses (see above) *A simple and reliable method is to mark respiration manually, i e watching the breathing, to turn a line up and down on the record in phase with respiration Ordinarily there are three phases of respiration* inspiration expiration and apnea (in this sequence) At more rapid rates of breathing the first two phases may not be interrupted by an apneic period

In the respirations trace in the recordings presented in this monograph *upward* movement of the line indicates *inspiration* a *downward* movement *expiration* and a *horizontal* position of the line apnea (The reader should not be confused by the fact that often inspiration has been marked "insp" at the peak (i e end) of inspiration and expiration marked "exp" at the end of expiration or early in the apneic period) *In many of the recordings electrocardiogram and respiratory trace have been combined* i e the base line of the EKG has been moved up and down to indicate respiratory phase

For purposes of special investigations other reference *recordings are used* such as intracardiac pressures obtained by cardiac catheterization (570, 571), apex beat (175 176, 828), electrokymogram (1257), and so on The apex beat has been useful in defining the electrical mechanical interval in mitral stenosis (see Fig 284) and the validity of the interval so indicated is confirmed by correlation with ventricular pressures (778) Correlation of murmur shape and intensity with the pressure gradient across valves is now possible in laboratories where right and left heart catheterizations *are being performed Correlation of* murmurs with flow, especially velocity of flow, would be of interest and probably will be possible in the not distant future

The "aspect ratio"—the ratio of the horizontal and vertical axes of the record—is an important feature in phonocardiography Einthoven introduced the paper speed of 25 mm per sec for electrocardiography this time base has continued to be "standard" for the EKG Phonocardiography requires a faster paper speed, i e , a time scale which is "spread out" more for optimum definition Flexibility in manipulation of the time scale is desirable at least in a research instrument, although it may not be essential to a clinical *tool In spectral phonocardiography,* as practiced by means of the original model one second was represented by about 44 mm on the horizontal axis This display although determined *fortuitously by the commercial design of* the sound spectrograph modified for phonocardiography proved satisfactory for routine work However, a flexible time base is being incorporated as a feature of recent models

RECORDING LOCI The standard areas for recording are as for auscultation *aortic* (second right intercostal space), *pulmonary* (second left intercostal space) *LLSB* (left lower sternal border, in the fourth interspace) and *apex* (region of apex beat or, if none is present the left midclavicular line in the fifth interspace) Simultaneous recording from multiple loci has been developed to a high degree by Leatham (839) The principal usefulness has been in demonstrating the normal constitution of the heart sounds in the several cardinal areas of auscultation and in aiding the identification of the origin of certain components of the heart sounds in pathologic conditions

Several areas for recording other than the cardinal areas of auscultation have been recom

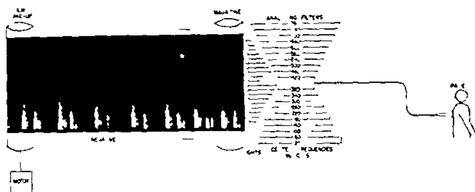


Fig 41A A highly schematic representation of the principle of a sound spectrograph which in this instance would provide direct (i e instantaneous) analysis and display (In the example of heart sounds used here the normal young subject demonstrates a third heart sound present only during inspiration There is also sinus arrhythmia)

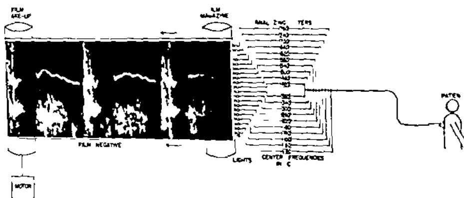


Fig 41B Same as Figure 41A except that lung sounds specifically from a patient with bronchial asthma are displayed Harmonics represented here as horizontal bands are the physical basis of the musicality of the sounds in asthma Note the much prolonged expiratory phase with a prominent harmonic

gist Since fetal phonocardiography is the study of heart sounds in the fetus spectral phonocardiography might indicate comparable studies in ghost The alternative designation spectrophonocardiography although possibly more accurate etymologically is too cumbersome for ordinary use

In effect the principle of the spectral analyzer is this (see Fig 41) One can think of this method as putting the sound (in electrical form of course) through a large number of electrical filters each of which is tuned to a different and specific frequency pass band The output of each filter could activate a tiny light The play of the bank of lights on the moving film would inscribe the spectral phonocardiogram Note that time is on the horizontal axis as in all physiologic recording The vertical axis is frequency scale not intensity as in the oscillographic recording Intensity in the SPCG is represented by density of a given area of the recording, for example in the schematic example used in this explanation note that the intensity of each of the tiny lights—and the density of the mark it makes on the recording medium—is a function of the energy in each frequency band of the sound being analyzed

In practice (see Fig 42) because of the expense and other difficulties of a large number of individual filters the method used is in general terms of this type The segment of sound which by aural editing is selected for analysis and display is played over from the original tape to the magnetic margin of a disk mounted on the same axis as the kymograph drum on which the records are made The analyzer itself is in essence a single pass-band filter the tuning of which is changed

catheter or on a rigid needle introduced directly into the chambers of the heart may, with care, provide more accurate information[7] At present there is no accurate information on the magnitude of the sound pressures in the heart The information in the next paragraph must be interpreted cautiously in light of these numerous difficulties

Intracardiac phonocardiography, like esophageal phonocardiography, demonstrates an atrial heart sound in all cases unless atrial systole is absent It is recorded loudest in the atrium and less clearly in the ventricle There is a striking restriction of sound to the vessel or chamber in which it is generated Recordings of the left side of the heart have been made in connection with "left heart catheterization" There is as much as a 100 fold attenuation of sound generated in one ventricle and detected in the other and an attenuation in excess of 60 db between the interior of the heart and the surface of the chest The first sound is much less intense in the vicinity of the tricuspid valve than in the vicinity of the interventricular septum (895) suggesting that the tricuspid valve may be a poor noise maker What can be interpreted as a physiologic opening snap of the AV valves can be recorded from the interior of the heart (979) In the first portion of the pulmonary artery (but not the aorta) a systolic murmur is recorded in all cases It is possible that a systolic murmur is, indeed, always present in this vessel at the peak of systolic ejection In direct phonocardiography a systolic murmur is always found over the base of the pulmonary artery and never over the aorta (1307) a corroborative finding However there is the additional possibility—in fact virtual certainty—that the presence of the catheter favors the development of a murmur because on fluoroscopy violent whipping about of the catheter is seen Often when the catheter microphone has been passed into the pulmonary artery or aorta from the ventricle a faint decrescendo diastolic murmur is recorded Presumably this is caused by regurgitation at the arterial valve which is held open by the catheter The murmur of mitral regurgitation is loudest in the left atrium The murmur of mitral stenosis is loudest in the left ventricle

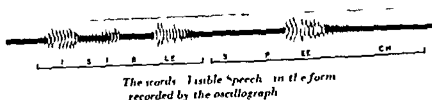
[7] Careful investigation of the frequency characteristics of catheter recording systems such as those by the Copenhagen group (636 637 1507) are pertinent

Potentially intracardiac phonocardiography will teach us much about the sounds we hear or record at the surface of the chest However, it is unlikely to become a routine procedure, even in cardiac catheterization laboratories, simply because it is unlikely that the information afforded will be sufficiently superior to that obtainable at the surface of the chest to make the increased risks and technical difficulties worthwhile

Rushmer and colleagues (1325, 1328 1329) have developed a technique for producing what they call *sonvelograms* In essence these are the *intensity envelope of the sound* This is accomplished, first, by half wave rectification of the oscillographic phonocardiogram, and second, by electrical integration The resulting curves can be used for timing purposes and possibly the shape of murmurs will be useful in making hemodynamic correlations

Spectral phonocardiography (Fig 41 to 43) is the application to phonocardiography of the method of sound spectrography developed at the Bell Telephone Laboratories in the early 1940's As in the case of all phonocardiography a display of the heart sounds is provided The record can be either direct written (on electrosensitive paper) or photographic The initial recording of the heart sounds together with reference recording is made precisely as for conventional phonocardiography, that is the recording can be made directly to the magnetic recorder of the display unit or and this is for several reasons the preferred practice the information can be first recorded on magnetic tape There is then an additional step in spectral phonocardiography between the recording of the sounds and inscription of the display the frequency analysis[8] The finished spectrogram is a time frequency intensity plot The analysis to which the sounds are subjected is in many ways comparable to that which the ear makes This method is called spectral phonocardiography because although it is a type of phonocardiography it differs from the conventional oscillographic variety in that the frequency spectrum of the sounds is portrayed "Spectral phonocardiography" may not completely satisfy the philolo

[8] The publications of Burger and colleagues (195 196) illustrate the more laborious and non graphic methods of frequency analysis—that of Fourier and that of Labrouste



The words Visible Speech in the form recorded by the oscillograph



The words "Visible Speech" as recorded by the sound spectrograph

Fig 43 From Potter et al (1230)

ferred bec au e of (1) flexibility in manipulation of the time c ale and (2) good den ity detail for represent ition of inten ity

Another filter s tem u ing a principle di tinct from the heterodyne method ha been devi ed by Huggin —the pha e filter (722 1514) Al though thi re ults in improved definition and more accurate repre entation of the harmonic of mu ical murmur (Fig 43) noisy ound which represent the majority of those generated in the cardiova scular s tem are demonstrated le s well in our opinion Tran ients e g the heart ound) are poorly defined in the phase filter record

In the preface (p vii) cert un of the virtues of pectral phonocardiographs were enumerated Figure 44 contra ts the o cillogram and the pectrogram of peech ound There i ufficient additional detail in the pectrogram to permit one with training to read what is being said This i of course impos ible from the o cillo gram

In it application to cardiovascular sound sound pectrography required everal methodo logic adapt ation (1) Empiric determination of the mo t satisfactory width of the frequency band for individual scanning and (2) devi ing

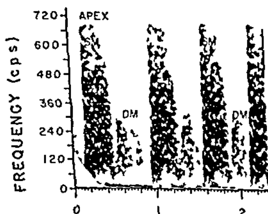


Fig 43 1 Recording of a noi y murmur made with the pha e filter (1101)

of method for incorporating the electrocardio gram and other phy iologic events in the fini hed record Other problems included determination of the be t frequency span for routine use and the be t a pect ratio i e ratio of vertical to hori zontal axis (see p 95) Al o the que tion of whether a logarithmic frequency scale might be advantageous required an an wer

With the wide pas bands there is abrupt and early onset and a minimum of ringing o that

FIG 42 1 Schematic repre entation of the sound spectrograph used in making most of the spectral phonocardio grams pre ented in this monograph The intermediate step of tape recording and the seriatim rather than simul taneous frequency analysis are shown

progressively as the segment of sound is played back repeatedly through it For example during the first rotation of the magnetic disk and the coaxial drum, the information in the 15 to 20 c p s band might be inscribed on the record with the second rotation the information in the 20 to 25 c p s band, and so on from bottom to top of the record until the entirety of the frequency scale desired has been scanned Actually there is much overlap of the individual frequency bands In making an analysis from 0 to 750 c p s the sound may be passed through the equivalent of as many as 425 individual filters each with its pass band overlapping others but with its center frequency about 1 8 c p s removed from the center frequency of the filter above and below (see Fig 43) It must be granted that the use of a single variable filter has the theoretical disad vantage that the Q or characteristic with respect *to ringing varies with frequency The temporal* error increases at higher frequencies This factor is, however of doubtful significance in spectral phonocardiography

The filter set up is of the heterodyne type and can be compared to the tuning arrangement of the ordinary radio, which also uses the heterodyne *principle in turning* the dial for tuning in' stations one changes the tuning of the oscillator in the radio set When the oscillator is in perfect resonance with a particular frequency the sounds at that frequency are picked up Only a station

FIG 42*B* Schematic repre entation of spectral phono cardiography at stage of double di play unit The heart ounds are fir t recorded on magnetic tape EKG res piratory pha e and other physiologic events are re corded imultaneously on tape by means of frequency modulated carriers The tapes are sub equently audited and ections elected for analysi At the stage of evolu tion of the analyzer and display unit represented here simultaneous direct written *(below)* and photographic *(above)* records were made

operating in the same frequency band as that at which the set is tuned will come through clearly

Note that the information can be put on a rec ord in several ways (Fig 43) The output of the filter can be used to activate an electric spark which marks electrosensitive paper Electrosensi tive paper has the disadvantage of a rather poor intensity depth Or the output can modulate a tiny light which is made to play on photographic paper Finally the output can be put into a cathode ray oscilloscope with a long persistence face and the image photographed The last method is pre

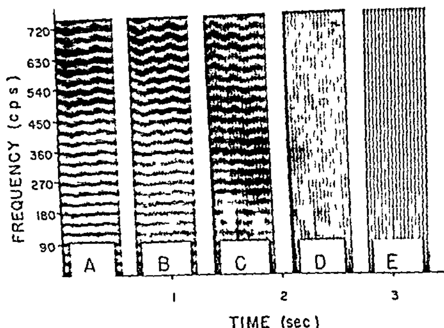


Fig 46 Analysis through the five filter set ups characterized in Figure 45 of a signal with a repetition rate of 30×/sec and harmonics at each 30 c p s Note that pass band C provides best compromise resolution of both time and frequency (Note that the artifact— wow or wobble —due to irregular rotation of the drum becomes more striking at higher levels of frequency because the excursion of wobble is a percentage of the frequency at a given level)

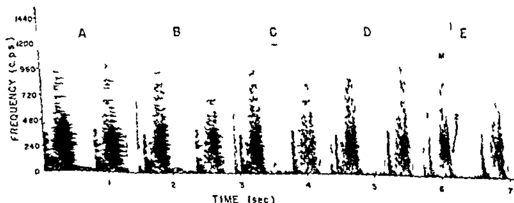


Fig 47 Crescendo or late systolic murmur displayed by the five filter system Pass band C is considered the best compromise for routine use in the study of cardiovascular sound

transients are sharply displayed but of course the frequency detail is by definition inferior With narrow pass bands the lag in activation tends to be greater and ringing occurs The records made with wide pass bands look streaked in the vertical direction those made with narrow filters look streaked in the horizontal direction because of the ringing (see Figures 45 to 48)

The instrument originally available to us commercially had a relatively wide and a relatively narrow filter system neither of which was satisfactory A filter of intermediate width (24 cycles wide at an attenuation of 6 db from peak performance) proved to give most satisfactory compromise definition of both time and frequency There is a difference in onset time of as much as

DIASTOLE
SYSTOLE
FREQUENCY (c p s)
600
450
300
150
0
0.5
1.0
TIME (sec)

FIG 44 *B* Recording of a musical murmur made with the phase filter The phase filter is unsatisfactory for the analysis of noisy murmurs It is superior to the heterodyne system for sharp definition of the harmonics of musical murmurs This superiority is of questionable practical value however

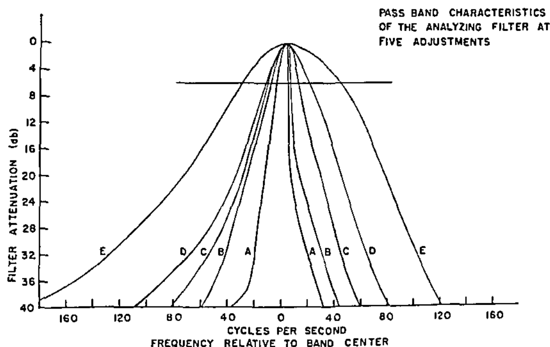


FIG 45 The pass band characteristics of five filter set ups tested to determine empirically the optimum system for study of heart sounds Defined in terms of width at 6 db attenuation (indicated by the horizontal line) these are A = 9 c p s B = 16 c p s C = 23 c p s D = 34 c p s and E = 71 c p s

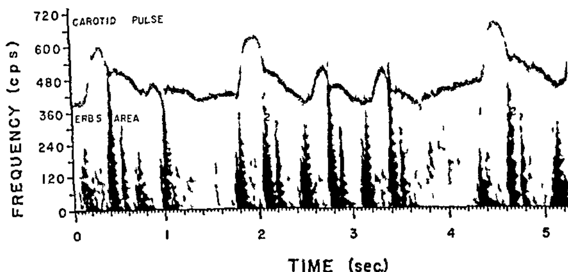


Fig. 50A Correlative recordings parallel with the spectrogram

Although the EKG was used in most of these studies (it was put on the tape as an FM signal and displayed directly on the final recording as part of the frequency analysis), any pressure pulse in electrical form can be similarly recorded and displayed, e.g., the carotid pulse (A) and the jugular venous pulse (B). C. B. (410809), a 57 year old man had chronic heart failure with atrial fibrillation. Three years previous to this recording a cerebral embolus occurred. The extra sound is probably third heart sound although it shows more variation with the duration of the previous diastolic interval than is usual. The alternative possibility is that it represents a mitral opening snap.

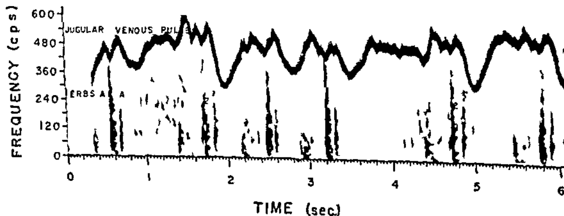
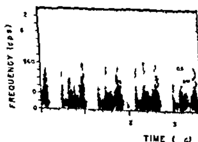


Fig. 50B See legend for Fig. 50A



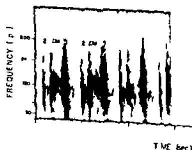


Fig. 51 Sound of mitral stenosis displayed with linear (*left*) and logarithmic (*right*) scales

0.015 sec between the widest and the narrowest filter systems (A and E in Fig 49) The lag in the filter system routinely used probably does not exceed 0.01 sec

The EKG and other low frequency physiologic data (Fig 50) are recorded on magnetic tape simultaneous with the initial sound recording This is accomplished by means of frequency modulated

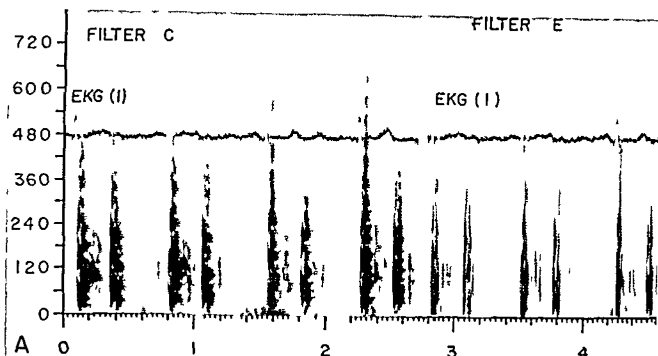


Fig 48 Pulmonary area in normal 14 year old patient The separate components of heart sound, specifically slight splitting, are best demonstrated by the wide filter system (E)

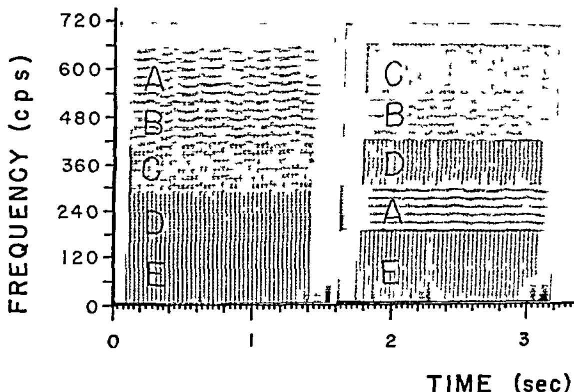


Fig 49 The relation of filter characteristics to time lag
The widest filter (E) has the least lag and the narrowest filter (A) has the most lag

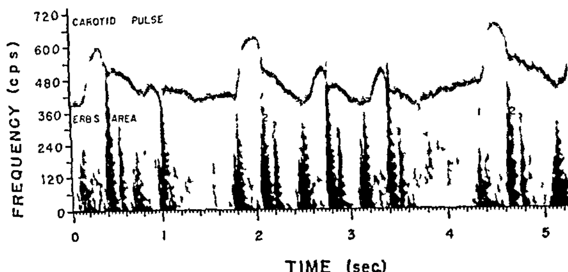


FIG. 50A. Correlative recordings parallel with the spectrogram.

Although the EKG was used in most of these studies (it was put on the tape as an FM signal and displayed directly on the final recording as part of the frequency analysis), any pressure pulse in electrical form can be similarly recorded and displayed, e.g., the carotid pulse (A) and the jugular venous pulse (B). C. B. (410300) [illegible] year old man has chronic heart failure with atrial fibrillation. Three years previous to this recording a cerebral embolus occurred. The extra sound is probably third heart sound although it shows more variation with the duration of the previous diastolic interval than is usual. The alternative possibility is that it represents a mitral opening snap.

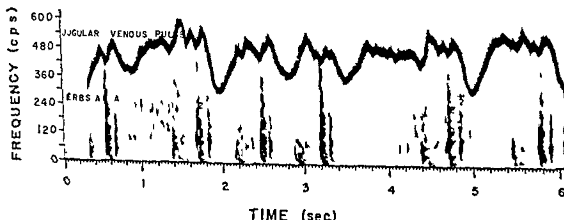
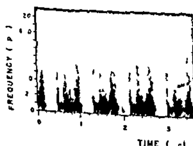


FIG. 50B. See legend for Fig. 50A.



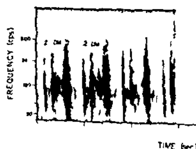


FIG. 51. Sounds of mitral stenosis displayed with linear (left) and logarithmic (right) scales.

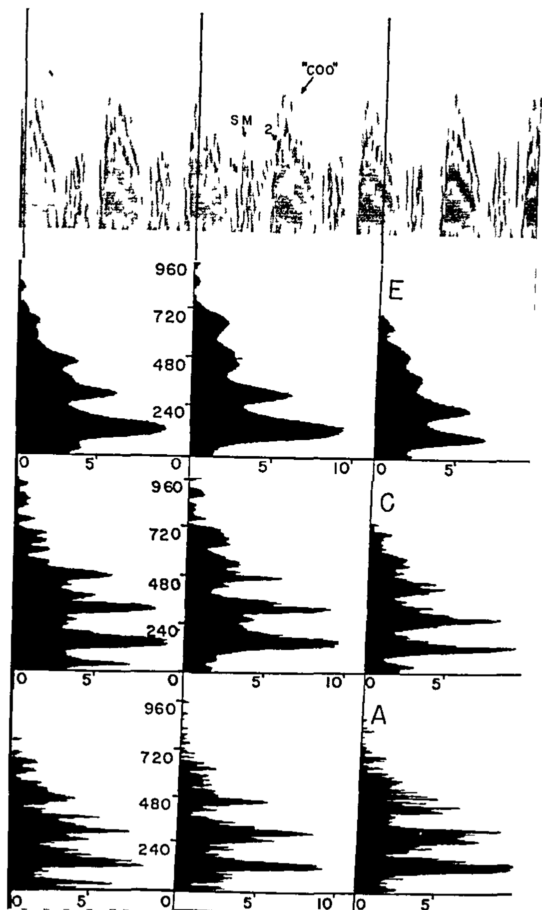


Fig 52 Intensity scanning

At the top is the spectral phonocardiogram of a "cooing" aortic diastolic murmur A systolic murmur is also present (filter I) Below are presented frequency × intensity sections taken at three specific points in the diastolic murmur (right to left) and analyzed through three filter systems which from above down are filters I, C and A

carriers For example the EKG may be put on at 3,000 c p.s The amplitude of the EKG is used to modulate the frequency level When this information which is in terms of frequency is put into the frequency analyzer an EKG or other curve of conventional appearance resulted By the use of frequency modulation one has in essence changed the electrocardiogram into a sound The same procedure is used in transmitted electrocardiograms over telephone lines but in that application demodulation has to be used to get the record back into conventional form (1240)

The rotation of the disk with the magnetic recording on its margin and of the kymographic drum which rotates on the same axis with the disk must be uniform Otherwise wobble or wow results For example if there is 5 per cent variation in the rate at which the drum rotates there will be 5 per cent variation in the frequency level of a constant pure tone In the case of low frequency sounds and particularly of noises the problem is of less importance than it is in connection with high frequency signals as the electrocardiogram In the EKG "wobble" shows up as bump which should not be there Only if they are very marked will they interfere with the QRS however they may render the P and T waves useless for correlative purposes

Preliminary surveys made it clear that a frequency span to 750 c p s would be adequate for the great majority of applications in cardiovascular sound This finding was *consistent with that of* Williams and Dodge (1566)

The aspect ratio available in the commercial model of sound spectrograph was satisfactory in our opinion This provided slightly less than 2 inches per sec on the time scale and 200 cycles *per inch on the* frequency scale The paper speed was approximately 44 mm per sec the standard EKG paper speed is 25 mm per sec

There are occasions when stretching of the time scale is desirable With the drum method this could be done only at the expense of the frequency display that is the change in the speed of the drum necessary to stretch out the time scale by a factor of 2 reduced the apparent frequencies (the height *of the* frequency scale) by a factor of 2 In brief the drum permits little latitude in the manipulation of the time base The cathode ray oscilloscope is a great improvement in this respect



Fig 53 A Three dimensional model

By cutting out and stacking a large number of intensity X frequency sections a three dimensional model can be constructed such as this one of the same case of aortic diastolic murmur as is shown in Figure 52 Note the curvature in the harmonics In general the results do not justify the effort

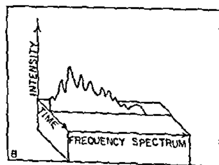


Fig 53 *B* Schematic drawing of Figure 53 A

Because most of the information in cardiovascular sound is under 200 c p s it seemed possible that a logarithmic scale (Fig 51) or at least a scale *with the* lower frequency range expanded would be valuable Cursory exploration of this possibility seemed to indicate that a logarithmic scale has no particular advantage However the analysis below 60–100 c p s leaves *much to be* desired since it is lacking in detail and the logarithmic scale by giving emphasis to this zone *of frequency actually* seems less informative than the linear *record It may* be premature to discard the expanded low frequency scale Improvement in the low frequency analysis is being worked out and a logarithmic scale may be considered optimum in the future

ARTIFACTS IN PHONOCARDIOGRAMS

PHYSIOLOGIC BUT NON-CARDIOVASCULAR NOISES (Fig 54 and 55) Breath sounds borborygmi and muscle noises are undoubtedly the principal inter

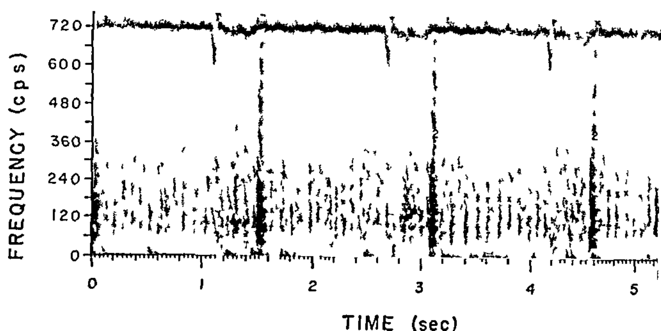


Fig 54 Muscle tremor

Repetitive transients due to muscle contractions are demonstrated in this recording from the aortic area. Two factors may result in such an artifact: (1) Straining by the patient in holding his breath; (2) shivering, often without the knowledge of the patient or physician, from cold room.

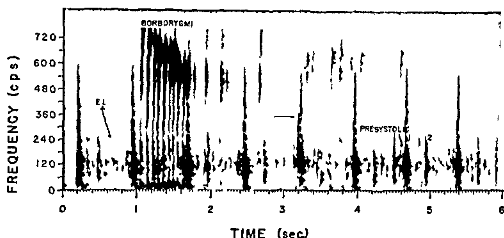


Fig 55 Artifacts in recording in apex in M M (242664) 38 year old patient with mitral stenosis. There are borborygmi—a series of rather musical transients—and electrical interference at 240 cycles. The sharp first sound and presystolic murmur are evident.

fering body noises. The breath sounds are more troublesome in the aortic and pulmonary areas, borborygmi at the apex. Muscle noises are mainly the result of shivering. *The patient may* himself not be aware of chilliness—insensible shivering. The room in which recording is done should be kept warmer than most rooms—certainly 70° F or higher.

In the oscillogram all artifacts are vibrations like cardiovascular sound. They may be difficult to differentiate from meaningful information of cardiovascular origin since the only means one has for identifying their artifactual nature is the lack of constant relationship to some element of *the electrocardiogram and the failure to recur* with each cardiac cycle. On the other hand, these body noises, as well as many of the major categories of ambient noises, have spectrographic characteristics which permit their identification in the SPCG.

The spectrographic characteristics of the breath *sounds are described in a later section (p. 487 ff).*

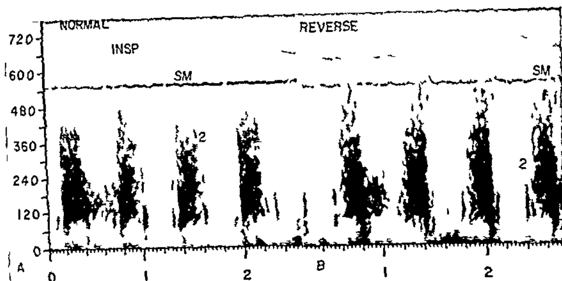


Fig. 54. A filter artifact

The recordings are from the apex of a patient with rheumatic mitral regurgitation. When analyzed in the customary fashion, the beginning of the murmur (A) was seen to have a suspiciously sharp front. It was suspected that a threshold characteristic of the filter system was responsible for such sharp front. The same segment of sound was played back in reverse into the analyzer. It was then seen that the sharp front was indeed largely artifact and that some detail was destroyed when the recording was made in the usual manner. This artifact, fortunately not too pronounced in the spectral phonocardiograph of current design, should be eliminated insofar as possible in future design.

Borborygmi (Fig. 55) occur as a series of transients which often have a rather musical quality. They may look rather like tympanitic percussion note (p. 475); the similarity is probably no accident. Muscle sound (Fig. 54) likewise present as a series of closely spaced transients. In children speech or other sounds of laryngeal origin may be a problem, but as in the case of ambient speech, sound is readily identified in the spectrogram by its harmonic pattern. The noise of snoring rarely is a problem; likewise displays harmonic pattern.

AMBIENT NOISE. The application of the microphone to the chest with air seal attenuates ambient noise by as much as 20 db. However, the phonocardiographer quickly becomes aware of the fact that the noise level in most laboratories is appreciably higher than one might at first guess. The ear quickly learns to disregard sound that is not pertinent. The oscillographic phonocardiograph cannot do that. Telephone bell and speech have a characteristic spectrographic appearance.

INSTRUMENTAL NOISE. Electrical interference is the major difficulty. Again this is more easily identified in the spectrogram since 60 cycle interference, for example, is seen as a line at 60 cycles.

ARTIFACTS UNIQUE TO SPECTRAL PHONOCARDIOGRAPHY. The sharp fronts, the introduction or rather display of high frequency component not truly there when the filter system is hit hard, false splitting of transients again when the filter system is overloaded—these artifacts unique to spectral phonocardiography are illustrated and discussed in Figures 56 to 58 and also in the Technical Appendix (p. 499).

In the oscillographic phonocardiogram time is on the horizontal axis and over-all intensity on the vertical axis. Predominant frequency is roughly indicated by the spacing of the oscillations; the more closely spaced are the vibrations, the higher is the dominant frequency. *In the spectral phonocardiogram time is the horizontal axis as in most physiologic recordings but the vertical axis (ordinate) is frequency scale in cycles per second, usually in a span from 0 to 750 c.p.s. Intensity at various frequency levels is indicated by degree of blackness (density) in the given portion of the recording.*

PCG is the abbreviation which will be used at times for *phonocardiogram* or *phonocardiography* or *phonocardiograph* (the context will make clear

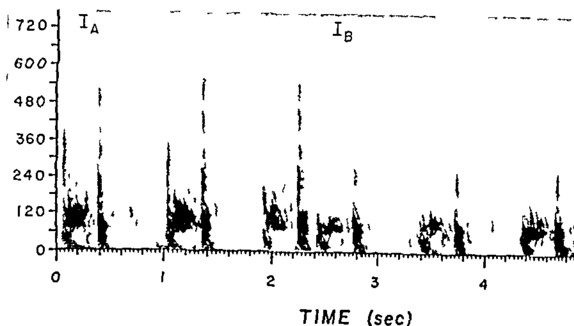


Fig. 57 I

Fig. 57. Another artifact of the filter system.

The experiment represented by these recordings was suggested by suspicions that artifactual high frequency components are added by the filter system in the case of sharp sounds of high intensity. Recording A in each pair of records was made in the usual manner. Recording B was made after passing the sound through a pass band filter which cut off components above 100 cycles. For analyses I, II and III the sound was put onto the magnetic recording disk of the analyzer at the same level of amplification but was played back during the analysis itself at three grades of amplification increasing from I to III. *For analyses IV, V and VI the sound was put through the analyzing* filter system with the same amplification but in recording onto the magnetic disk of the analyzer three grades of amplification were used, increasing from IV to VI. (The sounds are from the pulmonary area of a 15 year old boy with active rheumatic fever.)

The records demonstrate that, regardless of the stage at which additional amplification is applied, the analyzer displays frequencies appreciably above the level of cutoff of the pass band filter when the sound is very loud. Whether the components of higher frequency so displayed are truly artifactual depends on whether the pass band filter, which of course cannot be expected to cut off completely abruptly at 100 cycles, is likely to pass information up to 600 cycles, for instance, as in recording IIIB. Such is, in fact, impossible with the pass band filter employed, which produces an attenuation of 24 db per octave. This is, then, a filter artifact, but one which is not pronounced if overloading is avoided.

which is meant) and *SPCG* for the spectral equivalents. Sometimes *oscillogram* will be used for the conventional PCG and *spectrogram* for the SPCG.

The spectral phonocardiogram provides a visible representation of the same information which the ear derives from sound. There are theoretical reasons to anticipate superiority of the visual method for the study of cardiovascular sound. In evolution hearing developed later than vision (744). Vision is superior to audition in many respects. Pleasure, information and protection are more dependent on vision. Incidentally, as indicated earlier, the two do not blend well; sight competes seriously with hearing and usually takes the upper hand when the two senses operate simultaneously. These facts are related to the observations that blind persons are good musicians and that one closes his eyes to enjoy music to the fullest extent.

The SPCG closely resembles the mental image of heart sounds and murmurs. Potentially at least as much information can be displayed as can be made out by the auditory mechanism; still more can be displayed since the SPCG suffers from no physiologic masking, no fatigue phenomena and no peculiarities of intensity response at various frequency levels and since it has a time

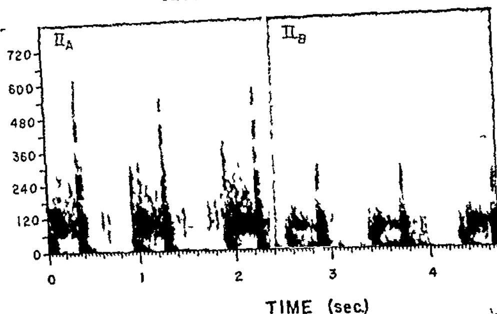


Fig. 57B

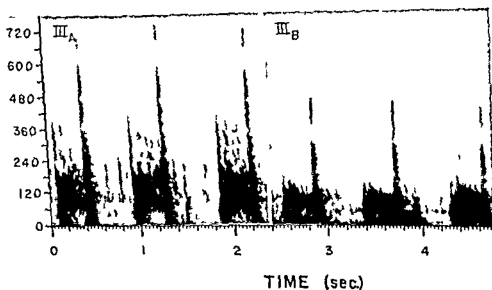


Fig. 57C

resolution superior to that of the ear. The improved resolution of time results from the spreading out of the frequency spectrum even if splitting is not discernible at points of higher intensity; it is likely to be evident in portions of the frequency scale where the split components are less intense. The spreading out of the frequency spectrum permits a display of the full dynamic range of cardiovascular sound; for example, a very faint diastolic murmur can be accurately represented in its true intensity proportions in the presence of a very loud systolic murmur (Fig. 59).

Since it emulates the ear, spectral phonocardiography is ideal for teaching clinical auscultation

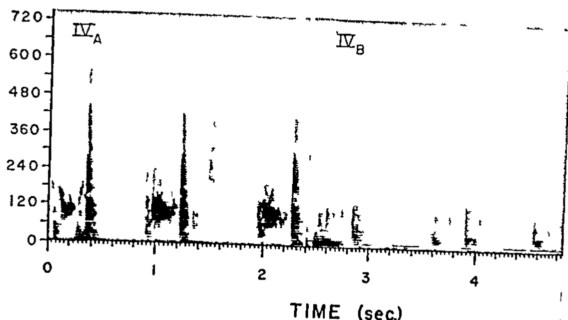


Fig 57D

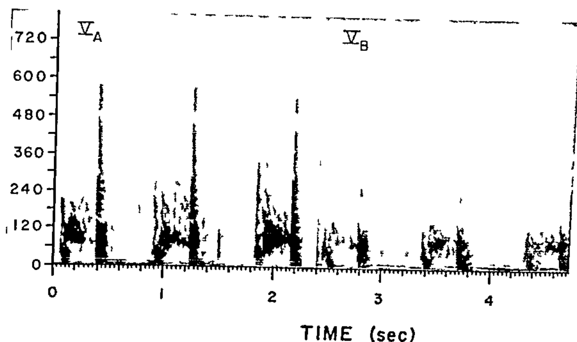


Fig 57F

for detailed presentation of medical sounds on the printed page and for precise quantifiable recording of the course of certain forms of heart disease and the effects of some types of cardiovascular surgery

Why do phonocardiography (1429)? The reasons are at least three

1 Phonocardiography has as one of its main functions to teach our ears what they should be hearing with the aid of the stethoscope It encourages self catechism during auscultation (442)

2 Phonocardiography provides valuable documentation particularly in the case of valvular heart disease bearing on the course of disease and the effect of cardiac surgery It keeps the clinician honest Dr William Dock makes the following

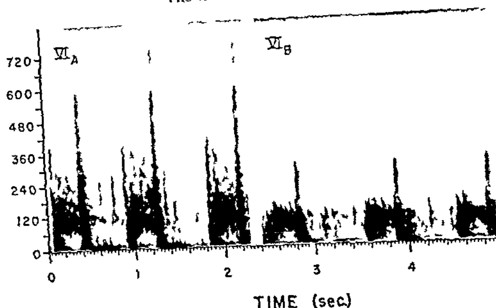


Fig 5 F

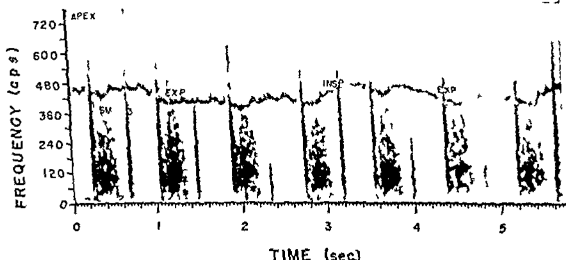


Fig 58 Microphone artifact from exce- ively inten e apex impul e

J Y (636 4) ag 16 years has rheumatic mitral regurgitation with moderate pulmonary hyperten ion by cardiac catheterization A forceful impul e wa felt at the time of the first heart ound and the third ound The napping soun l recorded immediately over the apex b at probably are artifactual in their appearance owing to overlo ding of micr phor e and amplifier The filter ystem of the pectrograph al o cannot be completely exonerate l (ee Fig 5) (See Fig 171 for split S in thi patient)

tatement One hould no more think of sending a patient for valve urgery without a phonocardi ogram than one would in the old day think of ending a patient to Trudeau without a che t X ray (See page 100 for a imilar quotation from the writing of Dock) Cardiac urgery—valvular urgery clo-ure of septal defect clo ure or creation of a ductus arterio u , etc —should be followed by pre and po t-operative recordings The evolution of heart disea e e pecially of valvular and congenital type is well followed by phonocardiography The following is another

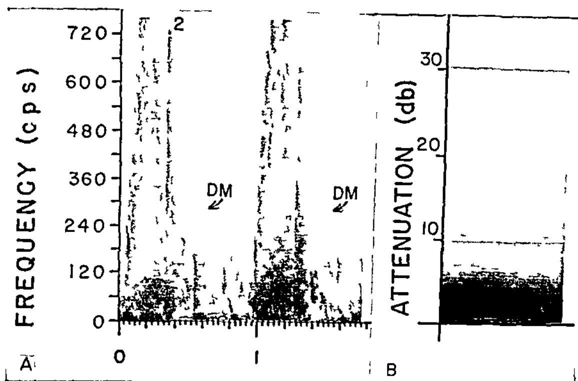


Fig 59 Display of large dynamic range in SPCG

In this case of rheumatic aortic stenosis and regurgitation the typical Christmas tree pattern of the murmur of aortic stenosis is displayed as well as the decrescendo pattern of the very faint diastolic murmur In the display of the wide dynamic range of cardiovascular sound spectral phonocardiography improves greatly on the performance of conventional oscillographic phonocardiography In the example given here (A) the systolic murmur is estimated to be about 60 db more intense than the diastolic murmur a 1000 fold difference There is no way by oscillography that these two murmurs can be simultaneously displayed in their true proportions In order to record the diastolic murmur with a maximum height of 3 0 mm the systolic murmur would have a height of approximately ten feet Using pass band filters will not solve the problem since the tremendous dynamic range is present at the frequency level where the diastolic murmur is most intense (about 280 c p s in this case) Use of *voltage limitation introduces distortion In its ability to encompass the large dynamic range of sound spectral* phonocardiography resembles the ear Technical developments in spectral phonocardiography must take this dynamic range into account In the tape recording maximum dynamic characteristics must be provided and the display medium must permit widest possible density grading from black to white In this regard photographic displays have a considerable advantage over the direct written record The display on electro sensitive paper provides a dynamic range of about 15 db (*B*) This last deficiency accounts for the homogeneous blackness of the lower frequency range of the systolic murmur in A The harmonic pattern of this portion of the murmur is completely obscured

quotation from Dock (3 7 p 64)

The permanent objective record supplied by phonocardiograms are as valuable in managing heart disease as blood smears in leukemia chest films in pulmonary tuberculosis or electrocardiograms in coronary disease Perhaps their greatest value is in teaching us to be better doctors at the bedside and in the office and by making us less prone to error when the phonocardiogram is not available

3 The phonocardiogram can provide information not available by ear for example (a) precise time measurements as in the case of the delayed first sound in mitral stenosis and the interval between the second sound and opening snap—measurements of quantitative significance in mitral stenosis (b) demonstration of low frequency sounds which are beyond the perception of the ear

And of course the ability to study the record at leisure and again and again is a considerable advantage Ability to represent the auditory impression on the printed page is an aid to education

and to scientific progress. Phonocardiography is especially useful when it is elevated from the domain of mere observation and used (1) for making semiquantitative physiologic estimates as of the level of left atrial pressure in mitral stenosis from the S_2-OS interval (p. 292) and of right ventricular pressure in pure pulmonary stenosis by the interval between the aortic and pulmonary components of the second sound (p. 382) and (2) in conjunction with physiologic and pharmacologic maneuvers designed to modify the heart sound (see Chapter 22).

The expense of phonocardiographic equipment and the complicated nature of the technique might make it seem doubtful that general clinical use will be forthcoming. However, it is not improbable that phonocardiography, specifically spectral phonocardiography (which will in my opinion become the method of choice) is now in a position comparable to that of electrocardiography about 1910. At that time there was expressed serious doubt that electrocardiography could ever prove clinically practicable. It is not inconceivable that the cumbersome research tool can be streamlined for clinical use just as was the electrocardiograph.

CHAPTER 8

The Structure and Function of the Heart Valves

The heart valves are intimately related to the fibrous skeleton of the heart, which is also the origin and insertion for the spiral myocardial bundles. The relationship of the four valves to the fibrous skeleton and to each other is demonstrated in Figure III 19 of reference 580.

The external topography of the heart valves was shown in Figure 37. In Figure 60 to 64 are presented radiographic demonstrations of the valve locations.

SEMILUNAR VALVES (Fig. 65). Since the mechanics of the semilunar (arterial, sigmoid) valves, aortic and pulmonary, is simpler than that of the atrioventricular (venous) valves, tricuspid and mitral, these will be discussed first. The cusps of the semilunar valves, normally three in number, are membranous sacs which in the position of closure fit snugly against each other providing mutual support. Each cusp has a thin area, the lunula, in which only minimal structural strength is present and necessary because of this mutual support. (Fenestrations occur most often perhaps in the lunulae (1051).) A fibrocartilaginous nodule, the corpus arantii, is located in the center of the margin of each cusp. Each of these nodules is so shaped that together the three fit to form a sphere.

Beyond the cusps the vessel is dilated into the sinuses of Valsalva. This feature has the effect of insuring that the cusps do not become plastered against the wall of the aortic root during ventricular ejection, a circumstance which would render subsequent closure of the valve difficult and might, furthermore, interfere with coronary flow.

There is confusion in the nomenclature of the heart valves (781). The one followed here seems the most easily remembered: the anteriorly located pulmonary valve has anterior, right and left cusps, the posteriorly located aortic valve has right coronary, left coronary, and posterior noncoronary cusps (see Fig. 66).

The opening of the semilunar valves is a simple matter of the pressure in the ventricle exceeding that in the aorta, is the result of ventricular contraction. Cinephotographs of the function of postmortem aortic or pulmonary valves, with ventricular systole simulated by a pump, provide an accurate picture, since there is little evidence that anything but hemodynamic factors, reproducible in the model, are involved. Such studies indicate that the valves are not completely open during systole but rather maintain an intermediate position. This tendency for a flap to be drawn toward the central axis of the stream of flow can be demonstrated by blowing across a sheet of paper which is held by its proximal edge (Fig. 67). The distal part of the paper which hangs down will be elevated, or if the paper is hung completely free, one may be able to raise it by a jet of air blown across it. Bernoulli's principle is doubtless operating in these situations, including that of the heart valves.

As was known to Leonardo, the aortic orifice is triangular (*cf.* Fig. 68) when the valve is open as a result of the intermediate position of each cusp. Quain (1236) gives the cross-sectional area of the base of the aorta as about 5.3 sq. cm., and McMillan (1088) estimates the area of the triangular orifice as 2.6–3.5 sq. cm.

Closure of the heart valves occurs with virtually no regurgitation—for practical purposes none. For example, in normal subjects no regurgitation into the left ventricle can be demon



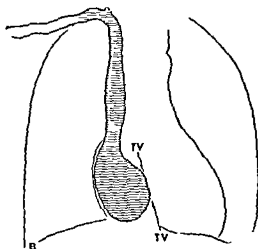
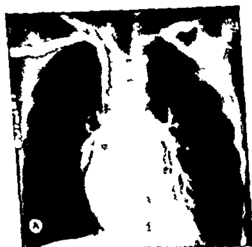


Fig 60 Angiocardiographic demonstration of the location of the tricuspid valve (TV) (Courtesy of Cooley and Sloan (291))



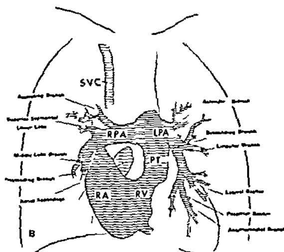


Fig 61 Angiocardiographic demonstration of location of pulmonary valve (between RV right ventricle and PT main pulmonary artery) Bulging of the pulmonary sinuses of Valsalva is demonstrated (Courtesy of Cooley and Sloan (291))

trated on aortography A pulling in of the cusps in the wake of the ejection jet places the cusps in an optimum position for efficient closure (670) The Leonardo da Vinci theory (see p 36) that vortices form on the outer aspect of the cusps in the sinuses of Valsalva and that when ventricular ejection slows and stops these uncoil like tightly wound watch springs and push the cusps shut seems unlikely although as recently as 1927 Hochrein (693) espoused this view Ceradini Luciani Henderson and others (see p 38) were of the opinion that breaking of the jet was the most important factor in closure of the arterial valve The ejection stream was visualized as a bolus of blood whose momentum carried it on briefly even after contraction of the ventricle ceased The arterial cusps were thought to be drawn in in the wake of the jet (See Fig 69)

The mechanics of bicuspid and quadricuspid valves (see p 37 for some historical details) and the phenomenon of trigonoidation may be discussed at this point especially since these are

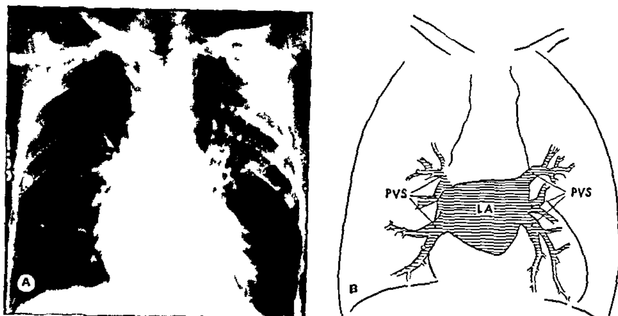


Fig. 62 Angiocardiographic demonstration of the location of the mitral valve. The mitral ring is represented by the somewhat flattened left contour of the left atrium. (Courtesy of Cooley and Sloan (291).)



Fig. 63 Rheumatic aortic stenosis with calcification (arrows) of the aortic valve. This 23 year old male had syncopal attacks, one of which was fatal. The heart shows surprisingly little enlargement. *A* Conventional film. *B* Heavy exposure at one-sixtieth second to improve demonstration of the aortic valve calcification visible by fluoroscopy. Autopsy confirmed the radiologic diagnosis of aortic valve calcification. (Courtesy of Cooley and Sloan (291).)

biological variants of the normal situation and perhaps cannot be considered strictly abnormal. The bicuspid aortic valve cannot open efficiently and tends to produce some degree of obstacle to forward flow through the orifice. The fact that slight regurgitation often develops at bicuspid valves suggests that closure in these valves may not be as reliable as in the tricuspid valve; however, it is more likely that the regurgitation is the result of the secondary atherosclerotic change to which these improperly engineered valves are prone and of the hypertension of coarctation of the aorta with which bicuspid aortic valve is often associated.

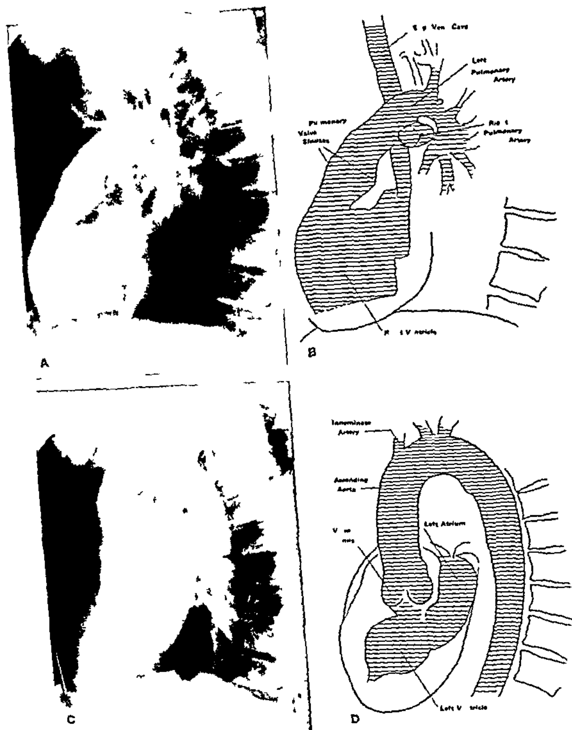


FIG 64 Angiocardiographic demonstration of the location of the pulmonary (A and B) and aortic (C and D) valves Lateral views (Courtesy of Cooley and Sloan (90))

Quadricuspid valves have an unfavorable ratio between the apex base dimension and the base dimension Whereas the bicuspid valve functions less than optimally in opening a quadricuspid valve is relatively weak in performing the function of guarding the mouth of the great artery during ventricular diastole

Quadricuspid valves occur more commonly at



Fig 65 Functioning heart valves

In a and b are presented successive frames from a motion picture (made at the speed of 24 frames per second) of the closing of the pulmonary valve in an isolated surviving beef heart In *c* to *f* are presented comparable series *in the closure of the tricuspid valve in the same heart The tricuspid valve took about twice as long to close* The tricuspid valve closed first at the middle Faster closure of the arterial valves may be in part responsible for the *fact* that the second heart sound is usually shorter with more high frequency components than the first (From film PMF 5162 made by the Armed Forces Institute of Pathology)

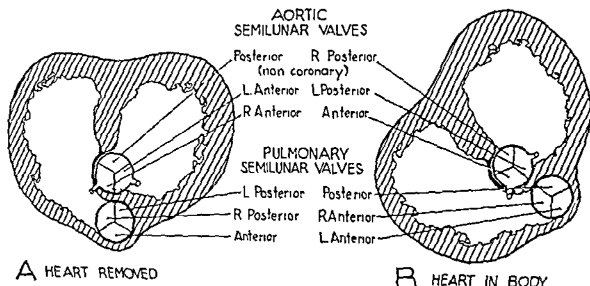


FIG 66 The nomenclature of the aortic and pulmonary cusps (From Gould (480).) The system indicated in the left hand column is the one which has been used in this monograph



FIG [illegible] Blowing across paper provides a graphic classroom demonstration of why the arterial leaflets are held in a mid position during systolic ejection

the pulmonary orifice A quadricuspid pulmonary valve has been found in an incidence between about 0 04 and 0 25 per cent in a series of properly studied autopsies (798) Occasionally (798) pulmonary regurgitation occurs with it as was predicted by Leonardo da Vinci that it does not occur more often is probably related to the relatively low pressure in the pulmonary artery under normal circumstance

Bicuspid state occurs more often at the aortic valve Of all patients with coarctation of the aorta 20 to 40 per cent have bicuspid aortic valve These valves commonly undergo calcific atherosclerotic and even stenotic change and even more commonly display regurgitation in some degree Rheumatic fever may cause fusion of two aortic cusps with resulting simulation of a congenitally bicuspid valve

Chisholm (262) pointed out that what he dubbed trigonoidation is likely to occur in the case of the thin walled pulmonary artery especially under condition of high pulmonary flow The illustration (Figure 70) will aid in understanding the phenomenon The result is that during ejection of the right ventricle the pulmonary cusps are pulled taut across a sector of the valve orifice The portion of the free margin of the cusp stretched in this manner may function as a noise generator possibly being related particularly to the musical variety of so called functional murmur (see p 244) In connection with the concept of trigonoidation the following observations are of interest and possible pertinence when considered together (1) With a properly sensitive pick up device at least a faint systolic murmur can be demonstrated at the surface of the chest in all persons (610) (2) Intracardiac phonocardiograms demonstrate a systolic murmur in the pulmonary artery in all cases (896) (3)

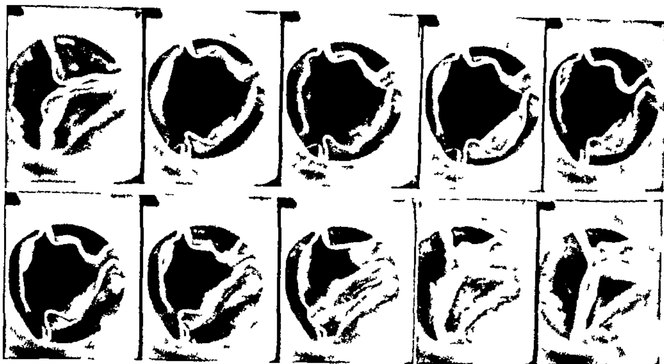


Fig 68 A cycle of normal aortic valve movements showing the opening and subsequent closure Successive cinematographic frames made with simulation of ventricular systole in a normal human heart by means of a pump (Courtesy of McMillan (1088) and the British Heart Journal)

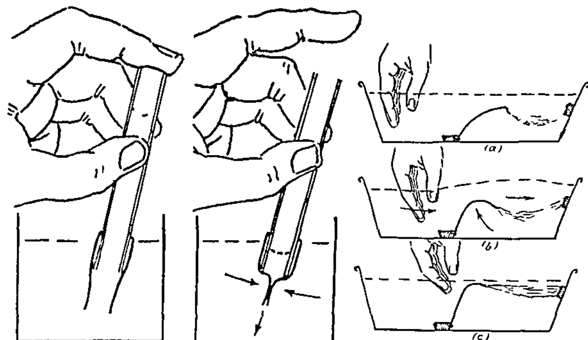


Fig 69 Demonstrations of the wake of the jet phenomenon (*Left*) Compare the situation schematized here with the atrioventricular valves or the arterial valve The fall of fluid in the tube is analogous to the propulsion of blood into the ventricle by atrial systole or into the aorta by ventricular systole Because of momentum the fluid falls below the general level of the fluid in the vessel and the flaps which are analogous to the valve cusps come together (*Right*) In the wake of a wave the flap is pulled in toward the center of the stream (From Henderson and Johnson (670))

Phonocardiograms recorded directly from the surface of the pulmonary artery in dogs and man demonstrate a systolic murmur in all cases (1307) (4) When phonocardiograms are taken directly from the surface of the pulmonary artery in dogs in which the pulmonary valve leaflets have been avulsed the systolic murmur disappears (1307)

The question of a role of the ventricular mus

lature in clo ure of the arterial valve i rai ed mainly in connection with ca es of ba ilar diastolic murmurs in association with ventricular failure (440) or with anemia and dilated ven tricle In brief the evidence i uch that an explanation for such a murmur mu t be ought elsewhere than in a dilatation and relaxation or weak contraction of the mu cular ring urrounding the aortic and pulmonary orifices In the ca e of the aortic orifice there is a separate myocardial bundle the deep bulbo piral (1002 1028 1281) which urround the outflow tract This mu cle contract of course during ventricular sy tole and probably ha no persi ting ten ion in early dia tole Therefore imperfect contraction would not be a likely factor in failure of the integrity of the aortic orifice Furthermore an atonic and dilated tate would probably not be a factor either although it i said that if the deep bulbo piral muscle i selectively cut in experimental animal aortic regurgitation occurs immediately [1] The fact that the aortic valve are water tight to the extent of with tanding very high pressures ap plied on the aortic ide in autop y heart i fur ther evidence agan t a mu cular element in arterial valve clo ure The aortic valve i part of the aorta not the ventricle and i not affected by dilation of the ventricle

Brock (179B) ha a sembled phyllogenetic anatomical and clinical data ugge ting that muscular function i important to the clo ure of the pulmonary valve He tate that the con clu ion that the control of the pulmonary out flow is a purely mechanical function of the valve cu ps and i not upported by mu cular action in the mammalian heart is almo t certainly in correct it i certainly incomplete It i plau ible to pre ume that trigonoidation (which results

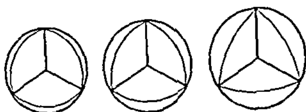




Fig 0 Trigonoidation of the pulmonary valve (*Above*) Schematic repre entation of succes ive de grees of dilatation and trigonoidation (*Below*) Photo graph of the pulmonary orifice in a necropsy specimen whi h wa fixed with a plug of cotton in the conus (From Chi holm (26?))

in not only a y tolic but also a diastolic murmur (330A)) would be more likely to occur if y tolic function of the infundibular mu culature were defective Pulmonary regurgitation may persi t for ome days after open direct surgery for Fallot s tetralogy (179B) Brock (179B) de scribes a patient who had had open repair of pure infundibular steno i and howed pulmonary regurgitation only during a ub equent preg nancy

THE ATRIOVENTRICULAR VALVES The mitral valve has two cu p (anterior septal or aortic po terior or mural) with chordae tendineae which attach to two papillary muscles The tricu pid valve which is attached by chordae tendineae to three papillary mu cles has two main cu ps (largest anterior infundibular smaller posterior

[1] In di cu ing the paper of Anders (19) Stewart referred to the exi tence of a mu cle pecifically sur rounding the outflow tract of the left ventricle and tated that if we expo e the heart of an animal and in ert a fine curved knife through the wall of the ventricle o as to divide the mu cle without injuring in any way the aortic cu p a dia tolic murmur of aortic in ufficiency will at once become audible There i a great need for a c reful and critical repeating of this experiment which I have found de cribed nowhere except in Stewart s di u ion taken down t nographically For th rea ons ent l above a mu cular fact r in aortic regurgitation is doubtful but the matter cannot be con idered ettled until Stewart experiment is carefully repeated



Fig 71 The fanning out of the collagen bundles of the chordae tendineae in the substance of the mitral cusp That each papillary muscle sends chordae tendineae to both cusps is evident (From Rushmer (1321))

inferior) and a smallest intermediate (septal) cusp The mechanics of the two AV valves have many identical features despite the variation in structure In fact the difference in structure is not so striking since the intermediate cusp of the tricuspid is often so small that the valve is essentially bicuspid and occasionally even the mitral valve may have a small third cusp When 'mitral valve' is used in the following discussion assume that both AV valves are meant

The orientation of the tricuspid inflow tract to the pulmonary outflow tract is such that an angle of about 60° is subtended by the two paths On the other hand the corresponding mitral and aortic paths on the left side are essentially parallel to each other (583) It has been thought that the

arrangement on the right side is more conducive to regurgitation at the AV valve than that on the left

The chordae tendineae are collagenous cords fundamentally identical to ordinary tendons as intimated by the name In the cusps the fibers of a given chorda fan out in the substance of the valve membrane An attractive fan tracery re revealed best on transillumination of the cusp (Figure 71) results A comparison with architectural fan tracery such as that in the cathedral at Wells England has been made (179) A point of possible functional significance is the fact that a given papillary muscle ends chordae to two cusps

Undoubtedly the most important factor in all valve closure is a change in polarity of the pressure differential across the valve orifice In the case of the atrioventricular valves the rise in ventricular pressure (through contraction of the ventricle) above that in the atrium is the most important factor in closure

Possible ancillary factors contributing to closure of the AV valves will be discussed in the following order

1 Contraction of the muscle surrounding the mitral ring reducing the cross sectional area of the orifice,

2 Contraction of the papillary muscle drawing the cusps together

3 Pulling in of the cusps in the wake of the jet produced by atrial systole (670)

4 Contraction of atrial musculature extending onto the AV valves

The Leonardo da Vinci mechanism discussed in connection with the semilunar valves is unlikely here as there

The evidence that contraction of the muscular ring may contribute to closure of the mitral orifice is two-fold Motion pictures of the ventricle laid open while still beating revealed shortening of the portion of the ventricle constituting the ring (1407) Motion pictures of the mitral valve of the beating heart photographed from the atrial aspect likewise show reduction of the gross orifice size with ventricular contraction (767) In the second place mitral regurgitation accompanies calcification of the annulus fibrosus mitralis even when the cusps and rest of the

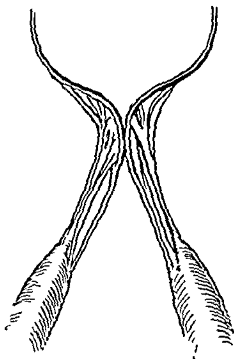


FIG 72 As is demonstrated in this simple sketch the atrioventricular valves probably appose over an appreciable portion of the margin This provides a certain margin of safety eg the cusps are sufficiently large that mitral regurgitation will not occur with slight or only moderate ventricular dilatation

valve mechanism are normal It has been suggested (1391) that the contraction of the muscular ring is frustrated in this condition However this situation is not entirely clarified Together the AV valve cusps have a surface area which exceeds the cross section area of the corresponding orifices (Fig 72) Probably the cusps will suffice to close the orifice even when the ventricle is considerably dilated The interior or aortic leaflet of the mitral valve can compensate for considerable deficiency of the posterior leaflet with the result that mitral regurgitation is unlikely to occur from lesions limited to the posterior cusp

Apposition of the cusps through contraction of the papillary muscle which end chordae to opposing cusps is inferred (1321) from the anatomy (Fig 71) each papillary muscle is connected by chordae to both leaves of the mitral valve for example The papillary muscles are thought to contract early because of their endocardial location Contraction of the papillary muscles, what

ever role it may have in the closure of the atrioventricular valve, is important in preventing partial eversion of the cusps into the atrium with ventricular systole and regurgitation is a result thereof. *Mitral regurgitation may develop from* improper function of the papillary muscle, for example from infarction subsequent to coronary occlusion, without actual rupture of the papillary *muscle or of the chordae. When the ventricle is* acutely dilated efficient closure of the AV valve may be impossible because the papillary muscle and chordae combination is too short. With marked dilatation of the ventricle the papillary muscles may become "absorbed" in the rest of the myocardium, that is, they are so stretched that they lose their separate identity.

In the closure of the arterial valves the "wake of the jet" mechanism is thought to be an important factor. That a similar mechanism may be operating in the case of the AV valves, with atrial systole being responsible for the jet, is suggested by several observations and experiments.

1. In dogs with sinus rhythm, injection of dye into the left ventricle reveals little or no evidence of mitral regurgitation. Appreciable regurgitation can be demonstrated if atrial fibrillation is induced (325). Henderson and Johnson (670) were inspired to investigate the "wake of the jet" mechanism by observing the remarkable ability *of the heart valves to close without attendant* regurgitation.

2. The length of the PR interval is one of the main factors in determining the intensity of the first heart sound. The degree of separation of the cusps of the AV valves at the onset of contraction of the ventricles is seemingly the immediate factor largely determining the intensity of the first sound. These considerations are pointed up by the chart presented in Figure 129. In a dog with surgically produced complete atrioventricular dissociation the intensity of the first sound was plotted against the length of the preceding PR interval. When *the atrium contracts before* the ventricle by an interval corresponding to a PR interval in the normal range the first sound shows minimal intensity. This is interpreted as *indicating* that the cusps are relatively close together and the assumed close position of the cusps can in turn be interpreted as evidence that they have been drawn together in the wake of the jet produced by the just preceding atrial systole. With shorter PR intervals than normal it is thought that the cusps are widely spread, and with longer ones than normal it is thought that the cusps have again had an opportunity to resume a neutral position after having been *drawn in in the wake of the preceding atrial jet.*

In man, a faint first heart sound occurs with prolongation of atrioventricular conduction, as in acute rheumatic fever. A variable first heart sound *occurs with the Wenckebach type of* second degree heart block and particularly with complete heart block. *Bruit de canon* was a term the French gave to the occasional booming first heart sound that occurred with complete atrioventricular dissociation.

3. There are suggestions from the right atrial pressure curves (832) and jugular venous pulses (435), in cases of complete heart block, that closure of the tricuspid valve occurs momentarily following atrial systole.

The above observations, which seem so well accounted for on the basis of the experiments of *Henderson and Johnson (670), are difficult to* reconcile with the report of Rushmer and colleagues (1326) that in the intact dog radiopaque markers placed on the mitral valve show little *movement during the cardiac cycle.* Current thinking relates the heart sounds more to a tensing of the belly of the valve cusps than to a collision of cusp margins. Is it not possible that Rushmer has studied mainly motion of the cusp margin and that considerable motion of the cusp belly with an rolling in the wake of the jet may occur independent of movement of the margin of the cusp?

In animals atrial myocardial fibers have been shown to extend into the AV valves. These are less striking in man than in lower animals. It is doubtful that they are of any functional significance, *in man at least.* Erlanger (432) described the beating of this musculature for some time in an excised animal heart and thought it might account for the partial closure of the AV valves in presystole as observed by Dean.

It will be clear from the above discussion that

it is impossible to reproduce the function of the AV valves in the necropsy heart in as complete detail as one can with the arterial valves. Although useful information can be gained from cinematographic studies of normal and diseased mitral valves in a pump model, the artificiality of the set up must be constantly borne in mind.

As pointed out by Talbot (1458), an important function of both the AV and the arterial valves may be to increase the efficiency of blood transfer. Both sets of valves funnel blood into the ventricle or aorta and tend to prevent the turbulent break up of the blood stream—a development which would lead to less efficient flow and to murmur production.

The ultrasonic Doppler method for demonstrating valvular function (1385A) and the correlations with cardiovascular sound promises to provide valuable information. It appears, for example, that opening of the normal AV valve is indicated in these recordings, as is also the movement accompanying atrial systole. Direct observation of these movements in the normal heart has hitherto been impossible.

CHAPTER 9

Temporal Relationship between Heart Sounds and Other Cardiovascular Phenomena

Basic to remembering the temporal relationships of *the first and second heart sounds* are the premises, now reasonably well established, that the first and second sounds are valve closure noises and that valve closure occurs, probably with some very slight lag, when there is a change in the polarity of the pressure differential across the valve orifice. Therefore, in simultaneous recordings of pressure on the two sides of the valve, the valve *will* open or close when the lines representing the respective pressures cross each other. As a generalization, too, it can be stated that valve opening is silent (see p. 12[illegible] for a discussion of this mooted matter). And it must of course be kept in mind that the first sound is a composite noise associated with closure of the *two* AV valves and the second sound a composite of the closure sounds of the *two* arterial valves.

The chart in Figure 73 presents information derived from cardiac catheterization and other sources. Closure of the aortic or pulmonary valve and the part of the second heart sound produced thereby corresponds to the incisura in the arterial pressure pulse of the corresponding great vessel. The closure of the AV valves and the resultant first heart sound occurs when the curve for ventricular pressure crosses that for pressure in the atrium.

When AV valve opening is productive of an opening snap, as in mitral stenosis, this sound occurs at the time that the ventricular pressure falls below atrial pressure. When the arterial valve opening is productive of an opening snap, as in pulmonic stenosis, the sound occurs at the time that ventricular pressure exceeds pulmonary arterial pressure.

The temporal relationship of events in the two ventricles and the effect this has on the relationship of the sounds generated on the two sides of the heart should be considered next. Again, cardiac catheterization has probably provided the most dependable information (166) (169). This information is reviewed in Figure 74 I. Contraction begins earlier in the left ventricle than in the right, with the result that the mitral valve tends to close slightly in advance of the tricuspid. Because of higher diastolic pressure in the aorta than in the pulmonary artery, isometric contraction is longer in the left ventricle than in the right. Ejection from the right ventricle begins before ejection from the left ventricle.

Normal asynchronism in the contraction of the two ventricles has been demonstrated in the past in both animals (770) and man (1576). In the last decade electrokymography has been applied to the problem. Comparing the onset of the major ascending limb of the carotid pulse with the corresponding point on the pulmonary artery electrokymogram, Ellinger and associates (423) in 68 normal young adults found that ejection from the left ventricle began first in 33 subjects by 0.01-0.05 sec., from the right ventricle first in 21 subjects by a comparable interval, and from both ventricles synchronously in the remaining 14 subjects. On the other hand, using a similar method Luisada and Fleischner (984) found right ventricular ejection had precedence in all of 8

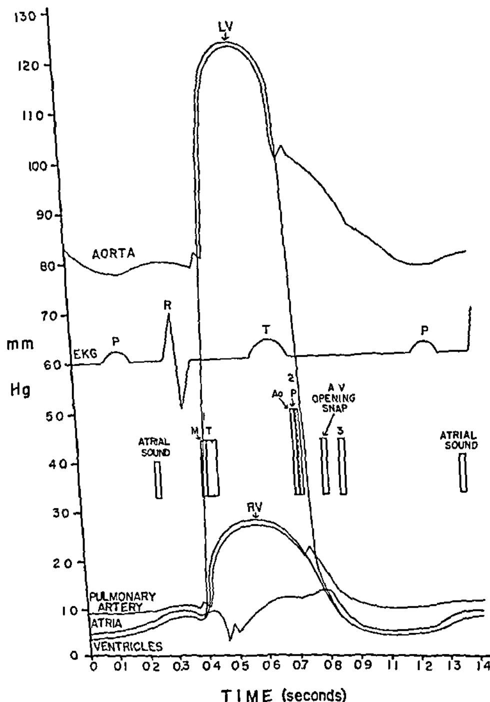


Fig. 73 Temporal relationship of the heart sounds

Shown are pressure curves in the aorta, left ventricle (LV), right ventricle (RV), pulmonary artery and atria (which for convenience are assumed to have identical curves of intracavitary pressure); also EKG. M, T, Ao and P refer to the closure sounds of mitral, tricuspid, aortic and pulmonic valves respectively. The intervals indicated by the time scale are not intended to be precise. A probable contribution to S produced when pressure in the left ventricle exceeds diastolic pressure in the aorta (and the aortic valve opens) is not indicated.

normal subjects by 0.025 to 0.03 sec Such demonstrations of asynchronism of the onset of ejection cannot be translated directly to time of closure of the atrioventricular valves *Because of* the higher diastolic pressure in the aorta than in the pulmonary artery *isometric contraction (after* closure of the AV valves) might be expected to be *longer in the left ventricle than in the right* By such a mechanism it is possible that mitral closure might slightly precede tricuspid closure and still *onset of ejection from the left ventricle* would occur later than that from the right Some studies indeed suggest that isometric contraction is longer in the left ventricle (On a rational basis, *precedence* of tricuspid closure over mitral closure *might be anticipated* since the right atrium contracts before the left, and by the time the ventricles contract the tricuspid leaflets have had time *to reach a slightly more closed position than* have the mitral leaflets)

Classically the cardiac cycle is variously divided into phases (1322) which are assigned different names according to the several nomenclatures *The following is an eclectic classification*

Iso(volu)metric contraction phase—between closure of AV valve and opening of arterial This interval has pertinence in connection with cardiovascular sound when there is an arterial opening snap, as in pulmonary stenosis or a systolic murmur generated at the arterial orifice because the interval between the first sound and the opening snap or beginning of the murmur is a measure of isometric contraction

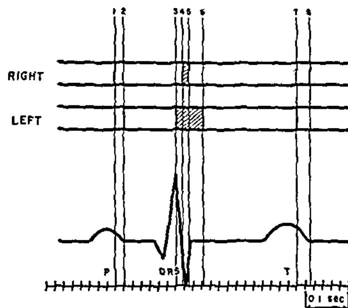


FIG 74A Diagrammatic representation of the average timing of electrical and mechanical events on both sides of the heart in normal subjects (1) onset of right atrial contraction (2) *onset of left atrial contraction* (3) onset of left ventricular contraction (4) onset of right *ventricular contraction (5) onset of right ventricular ejection* (6) onset of left ventricular ejection (7) *end of left ventricular ejection* (8) *end of right ventricular ejection*

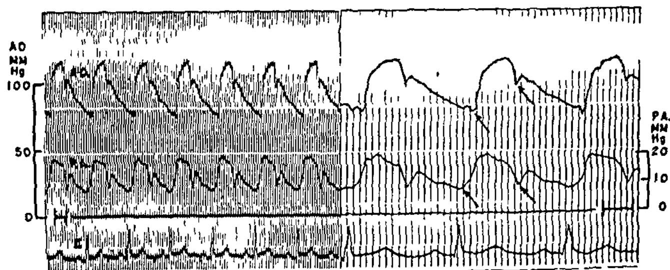


FIG 74B Simultaneous aortic (AO) and pulmonary artery (PA) pressure pulses in a subject with a normal circulation (illustrating the type of curves from which the information shown in A is derived) Note difference in time between corresponding events in the two curves Paper speed 25 mm / sec on left, 75 mm / sec on right Time lines 0.04 sec apart Arrows indicate beginning of ejection and end of ventricular systole (Courtesy of Braunwald, Fishman and Cournand (166) and *Circulation Research*)

Systolic ejection phase which on the basis of aortic pressure and/or ventricular volume curves is sometimes divided into phases of maximum ejection and reduced ejection (Theoretically at least there must be between the time that the ventricle begins to relax and the time of closure of the arterial valves a very brief period which is legitimately considered part of diastole and is indeed referred to as the protodiastolic period by Wiggers (1551) It is preferable to avoid this term because of the obvious possibility of a false association with protodiastolic gallop which occurs at quite a different part of the cardiac cycle)

Isometric relaxation phase—between closure of the arterial valve and opening of the AV valve This phase corresponds to the interval between the second sound and a mitral or tricuspid snap if present

Rapid filling phase

Reduced filling phase

Atrial systolic phase

It will be noted that there is no classically described phase for the interval between the onset of contraction in the ventricle and closure of the AV valve *Preisometric contraction phase* is a satisfactory designation (508 1139) This interval important in mitral (or tricuspid) stenosis has been referred to by Kelly as the *mechanical lag period* the interval between onset of the QRS of the electrocardiogram and onset of ventricular contraction being the *electrical lag period*

In making measurements in phonocardiograms the convention is to measure from the beginning of one component to the beginning of another For example the Q 1 interval is that separating the *beginning* of the QRS *from the beginning* of the rapid deflection or high frequency components of the first heart sound Splitting of S is measured in terms of the distance from the *beginning* of the first (usually aortic) component *to the beginning* of the second (usually *pulmonary*) component

The first sound—at least the portion caused by mitral closure—normally begins after the beginning of the QRS of the electrocardiogram by an interval which is about 0.05 sec for adults (875) and 0.05 second for children (1051) Strober et al (1449A) found in 100 subjects ages 2 to 35 years that the Q 1 interval varied between 0.02 and 0.06 sec with a mean of 0.04 sec

TABLE 2

Phonocardiographic time intervals in children †

Interval	N	M ± E(M)	S D
Q I	117	0 050 ± 0 001	0 015
II III	88	0 130 ± 0 003	0 024
I-IV	64	0 123 ± 0 003	0 021
IV-I	64	0 073 ± 0 003	0 023

N = Number of cases M ± E (M) = Mean ± standard error of the mean S D = Standard deviation

Children under 14 years equal sex distribution fairly *uniform age distribution*

† Modified from Table 18 p 9 of Mannheimer (1031)

The closest correlation of those analyzed was with height (r = 0 56 ± 0 11) The *coefficient* of correlation for weight *QRS duration age* and heart rate were 0 37, 0 20 0 12 and −0 22 respectively

The second sound bears no constant direct *relationship* to the *T* wave of the electrocardiogram except insofar as the interval between the first and second sounds is normally a measure of *mechanical systole* and mechanical systole bears a relationship to electrical systole *as measured by the QT interval*

Leonard Weissler and Warren (875) found a Q 1 interval (that between onset of QRS and first *rapid deflections attributed to mitral valve closure*) of 0 055 sec in normal subjects This figure accords well with that of Braunwald *et al* (166) who found an average interval of 0 052 sec between the onset of the QRS and the crossing of left ventricular and left atrial pressure *curves* This interval is prolonged in mitral stenosis (see p 286) because of elevation *of left atrial pressure* Curiously it is also *prolonged* to some extent (on an average (875) to about 0 070 sec) in systemic arterial hypertension See *p 427 for a discussion* of the basis of this delay

The interval between the first and second sounds is one of the more usable of the available indices of the *duration of mechanical systole* (1551) The length of systole varies with the heart rate as indicated in Figures 75 to 77 Increased activity of the sympathetic nervous system (1558) and situations such as thyrotoxicosis or pheochromocytoma in which an effective increase in sympathetic activity is produced by the humoral disorder of the disease cause abbreviation of systole out of

proportion to acceleration of heart rate. The curve for duration of mechanical systole in reference to heart rate is different in women and in men: for any given rate, systole is usually shorter in men (962), in all positions (upright or supine). The vagus nerve has only an indirect effect on the duration of ventricular contraction, i.e., through effects on heart rate. Mechanical systole is shorter in hypercalcemia and longer in hypocalcemia, although not as long as electrical systole. Changes in potassium and pH also affect the duration of systole. Cardi (234) found that with hypothermia electrical and mechanical systole are prolonged in a parallel fashion to a value three or four times the normal.

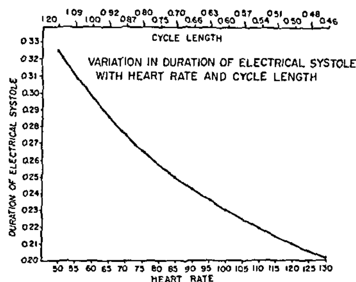


FIG. 75. The variation of electrical systole (QT duration) with heart rate (*below*) and cycle length (*above*).

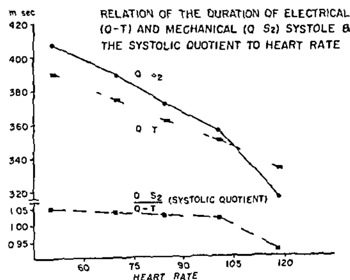


FIG. 76. The relation of electrical systole to mechanical systole. (From Kuhns (823).)

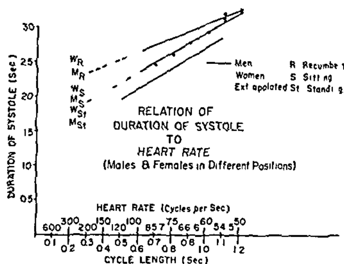


FIG. 77. The variation of mechanical systole (from arterial pulse wave) with heart rate, sex, and posture. From Lombard and Cope (962). The dashed segments of the line for male subjects were derived by extension of the rest of the line, which is based on actual observations in normal subjects. The data for females was less extensive than those for male. The formulae for the six straight lines are as follows (C = cycle length and S = systolic length in sec.):

W_R (women recumbent)	S = 0.065C + 0.24[illegible]
M_R (men recumbent)	S = 0.105C + 0.2010
W_S (women sitting)	S = 0.135C + 0.1683
M_S (men sitting)	S = 0.147C + 0.14[illegible]
W_{St} (women standing)	S = 0.157C + 0.317[illegible]
M_{St} (men standing)	S = 0.150C + 0.1242

Table 3 lists some factors responsible for shortening and lengthening of systole. Liberties have been taken in connection with several conditions since information is limited as far as mechanical systole is concerned and is much more ample for electrical systole because of the greater ease of measurement. Partial justification for judiciously using interchangeably information on the duration of mechanical and electrical systole is provided by Blair, Wedd and Young (111) who found that the duration of contraction is related linearly to the QT interval over a wide range. In man Cheer and Li (255) found reasonable correspondence of mechanical and electrical systole. The corroborative findings of Kuhns (823) in 271 students 20 to 30 years old are presented in Table 4 and Figure 76. Systolic quotient is the term he proposed for the ratio of mechanical systole to electrical systole.

As an exception to the relationship between mechanical and electrical systole, Hegglin (661) described a syndrome to which his name i

TABLE 3

*Factors affecting duration of mechanical systole**

Prolongation	Abbreviation
1 Female (962)	1 Male
2 Bradycardia	2 Tachycardia
3 Increased diastolic volume	3 Reduced diastolic volume e g in piration (LV)
a In piration (RV)	4 Thyrotoxicosis
b ASD (RV)	5 Epinephrine (1547 Fig 6) and sympathomimetic drugs
c PDA and VSD (LV)	6 Hypercalcemia
d After compensatory pause or extrasystole	7 Acidosis
e After saline infusion (154 Fig 6) (Luisada and Sarnoff)	8 Fever
f Elevation of legs (Weitz and Warnecke)	9 Digitalis (254)
g In complete AV block when PR interval appropriate (1389)	
4 Increased resistance to systolic ejection	
5 Hypocalcemia	
6 Alkalosis	
7 Hypothermia (234)	
8 Myxedema	
9 Quinine quinidine atabrine emetine	

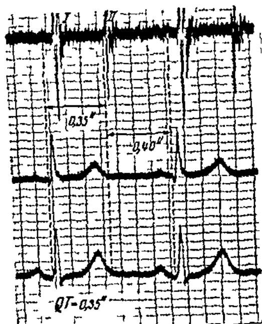
Since there is much more information on the duration of electrical systole than mechanical systole liberties have been taken in preparing this chart It is not proved in many cases that the same changes are seen in mechanical systole as in electrical systole

TABLE 4

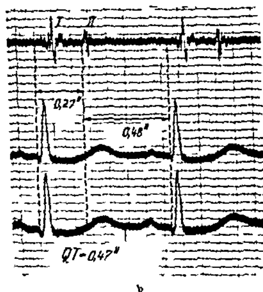
The relation of mechanical and electrical systol

Number of Subject	Heart Rate	Av Rate	QII (msec) with kma	QT (msec) with ecgm	Syst I Quo t (I II with gm)
5t	45-60	55	408±2	388±23	1 0 ±0 01
118	61 5	60	93±16	3 ±19	1 04±0 01
66	6-90	8	373±18	363±16	1 03±0 01
21	91 105	96	351±1	347±20	1 02±0 01
8	106-120	118	311±18	3 ±15	0 96±0 00

Data of Kuhn
Sigma = standard deviation



a



b

FIG 78 A Normal B Hegglin s syndrome Note that mechanical systole is shorter than normal and electrical systole is much longer than normal (From Hegglin (661))

sometimes is signed of energetic-dynamic circulatory insufficiency (Fig 78) The syndrome is characterized by mechanical systole shorter than electric systole (It is interesting that a converse situation occurs normally in the non hibernating hedgehog (748) and in the kangaroo (1426) mechanical systole is longer than electrical systole) Hegglin found his syndrome in association with diabetic coma and with liver disease Kuhns (823) points out that three combinations can lead to the situation of mechanical systole shorter than electrical systole

(1) Normal QS long QT
(2) short QS_1 normal QT
(3) short QS_1 long QT

The third situation is one to which Hegglin specifically referred as "energetisch dynamische Herzinsufficienz."

For electrical systole Bazett suggested the formula

$$K = \frac{QT}{\sqrt{\text{Cycle length (sec)}}}$$

where K is 0.37 for men and 0.4 for women. Ashman and Hull (32, 33) used the following formula: $QT = K \log_{10}(R - R + 0.07)$ in which K was *0.385 for males and 0.375 for females.*

The duration of mechanical ventricular systole in man was studied by Lombard and Cope (962), using the carotid pulse tracing which in fact is an index of ventricular ejection time rather than of *total contraction time. They emphasized the* large influence of the volume of venous inflow on the duration of ventricular systole and the negligible and inconsistent influence of arterial pressure (systolic pressure, diastolic pressure, or pulse pressure). They recognized that the heart sound method is probably the most accurate for determining this value. The duration of systolic ejection as measured by Lombard and Cope was shortest in the standing position, longer in the sitting, longest in the recumbent—the expected effects of difference in venous filling. Height, weight, age (15 to 65 years), season of the year, and use of tobacco had no apparent influence. Women had longer systoles, particularly for their rates. Because of beat to beat variability, which one would suspect was probably largely respiratory *in origin, it was found necessary to use the* average of at least 15 cycles.

Lombard and Cope (962) found that the relation of the length of systole to cycle length could be described by a straight line formula, but suspected that the formula does not apply at rates in excess of about 120 per minute. The formulas they derived were as follows (C = cycle length, S = length of systole):

Men,	standing at rest	$S = 0.150C + 0.1242$
	sitting	$S = 0.147C + 0.147[illegible]$
	recumbent	$S = 0.10[illegible]C + 0.2010$
Women,	standing at rest	$S = 0.157C + 0.1375$
	sitting	$S = 0.135C + 0.1683$
	recumbent	$S = 0.06[illegible]C + 0.2478$

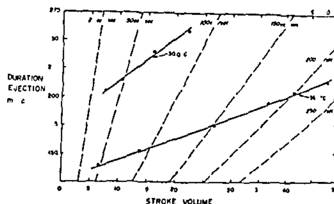


Fig. 79. Relationship between stroke volume and the *duration of systolic ejection per beat (as well as the consequent mean rate of ejection—dashed line.)* Heart rate constant at 115 per min. and mean aortic pressure constant at 100 mm. Hg in dog heart preparation of Sarnoff and collaborator (1318). Solid dots indicate values obtained at 36.2° C and open circles those at 30.0° C. *(Courtesy of Braunwald, Sarnoff and Stainsby* and of *Circulation Research* (171).)

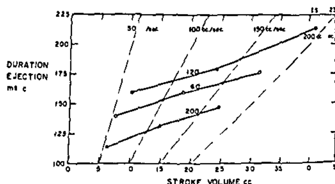


Fig. 80. Influence of heart rate on the relationship between stroke volume and systolic duration. Conditions otherwise same as in Figure 79. (Courtesy of Braunwald, Sarnoff and Stainsby and of *Circulation Research* (171).)

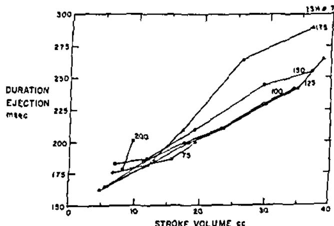


Fig. 81. Influence of aortic pressure on the relationship between stroke volume and duration of ejection. The numbers by each line indicate the pressures at which the values were obtained. (Courtesy of Braunwald, Sarnoff and Stainsby and of *Circulation Research* (171).)

Lian Golblin and Baruge (92a) used the formula

$$K = \frac{D}{C(C+41)}$$

in which D and C were diastolic period and complete cycle length in seconds respectively, as derived from the arterial pulse pressure curve. K was normally 0.004-0.0049. Long systoles gave values for K below 0.004, short systoles gave value above 0.0049. The values for C and D were obtained from the arterial pulse pressure curve.

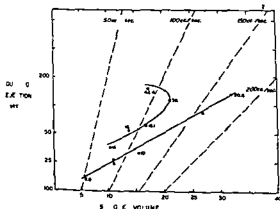


FIG 82 Relationship between stroke volume and duration of ejection (a) in the presence of a descending failure limb of the ventricular function curve (open circles) and (b) after the administration of 5 mg mephentermine sulfate (Wyamine) (solid dots). Numbers contiguous to the points indicate left ventricular end diastolic pressure in cm H_2O. (Courtesy of Braunwald, Sarnoff and Stainsby and of *Circulation Research* (171).)

TABLE 5

The hemodynamic determinants of systolic duration

	Duration of Ejection per Beat	Duration of Ejection per M	Mean Rate of Ejection
Increased stroke volume	↑	↑	↑
Failure limb of Starling curve	↑	↓	↑
Increased heart rate with constant stroke volume	↓	↑	↑
Increased heart rate with constant cardiac output	↓	↑	↓
Hypothermia	↑	↑	↓
Sympathomimetic amine	↓	↓	↑

From Braunwald, Sarnoff and Stainsby (171)

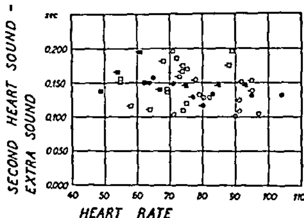


FIG 83 Relation of S_1-S_2 to heart rate

Squares male. Circles female. Physiologic sounds in black, pathologic open. (From Frost (498).) There is a slight trend toward a shorter interval at higher heart rates, a finding consistent with that shown in the next figure.

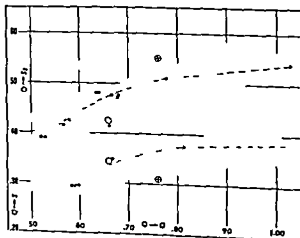


FIG 84 Relation of QS_2 and QS intervals to cycle length

Data from 70 cases of normal and diseased hearts in persons from the ages of 2 to 70 years. The large open circles are measurements in a patient with constrictive pericarditis before surgery and the large crossed circles after surgery. The chart indicates the fact that before operation the protodiastolic sound was closer to S_2 than in the average case and that the interval increased after operation. It is of interest that systole was abbreviated after surgery. (From Dunn (387).)

Mechanical systole as measured by the heart sounds is normally very slightly shorter than electrical systole as gauged by the QT interval. Mannheimer (1031) found that electrical systole was 2.1 per cent longer in normal children and 3.4 per cent longer in children with congenital heart disease.

There is much clinical and experimental evidence that venous filling has an important effect on systolic duration. Systole is shorter when the subject is in the standing position. The duration of systole in each ventricle can vary independently of that in the other ventricle. With inspiration the duration is increased in the right ventricle and decreased in the left—changes which parallel those in venous filling. In atrial septal defect systolic duration is usually consistently longer in the right ventricle; in patent ductus arteriosus the left ventricle tends to have a longer systolic period. There is lengthening of the systolic period in the first normal beat after the compensatory pause of an extrasystole. Systole is longer with the intravenous infusion of saline and is briefer than is normal for the rate during shock. The lengthening of systole at slow heart rates, in complete heart block for example, is probably related to the increased stroke volume: the enlarged heart and large stroke volume of complete heart block indicates increased diastolic filling of the ventricle.

In a stable dog preparation (1348) in which they could vary venous filling Braunwald, Sarnoff and Stainsby (164, 171) found a linear relationship between stroke volume and duration of systole. See Figures 79 to 82 and Table 5. Increased mean aortic pressure had little effect until high levels were attained. At pressures approaching 200 mm Hg a prolongation of systole was observed. This finding is in disagreement with that of Wiggers (1553) who found an abbreviation of systole with acute elevations of aortic pressure.

In heart failure digitalis, which reduces diastolic volume of the heart, reduces the duration of systole (254) despite the lengthening that might be expected from the bradycardiac effect of this drug. In mitral stenosis Wells (1527) claims that ventricular systole is abnormally short because of reduced stroke volume. (Of course, the delay in the mitral first sound (see p. 286) causes the systole to be even shorter.)

The physiologic *third sound* occurs normally about 0.14 sec. after the beginning of the second heart sound. Frost (498) found in adults a mean value of 0.147 sec. (S.D. = 0.025). Mannheimer (1031) found shorter values in children (see Table 2). It occurs during the phase of rapid ventricular filling as indicated by the downstroke beyond the V peak of the venous pulse (the Y limb). One study found a seeming temporal relationship between S_3 and the peak of the V wave of the jugular venous pulse and interpreted this as indicating that S_3 is an opening sound of the AV valves. The temporal relationship is probably spurious and the result of time lag in the jugular venous pulse wave (828). Frost concluded that the interval between S_2 and S_3 is not influenced by heart rate or sex (497, 498) but does vary with age (983)—probably as a function of heart size—becoming longer at older ages (Fig. 83). Dunn (382) presents evidence for a change in S_2-S_3 interval with heart rate (Fig. 84). A systematic study of the relation of this interval to the length of the preceding diastolic period in sinus arrhythmia or in atrial fibrillation remains to be done.

The atrial heart sound usually occurs about 0.12 to 0.17 sec. after the beginning of the P wave (Frost (498) found a mean value of 0.141, σ = 0.023) and 0.05 to 0.09 sec. after the onset of atrial systole as signalled by the rise of intra-atrial pressure in the cardiac catheter curve. Cournand (310) found in eight adults an average latent period of 0.11 sec. between the onset of the P wave and the onset of the right atrial pressure wave.

CHAPTER 10

The Generation of Sound in the Cardiovascular System

Although it is occasionally difficult to draw the line between the two, cardiovascular sound can be divided into (1) circumscribed sounds or transients (actually complexes of transients in some cases): the heart sounds; and (2) longer combinations of vibrations usually called murmurs (or occasionally, especially in the case of peripheral vascular murmurs, *bruits* or *souffles*).

Mechanism is a flexible term and concept, like the word *basic*, which is sometimes combined with it. In the following discussion, when it is stated that a given sound is due to valve closure or that a given murmur is due to retroversion of a valve cusp, it is recognized that it may be more exact to state that sound is associated with a particular event or anatomic setup. It is recognized that when the mechanism of a sound is valve closure, the details of mechanism of the generation of the sound have not been elucidated.

TRANSIENTS

The first and second heart sounds are in the main valve closure noises (309, 910). The first sound is associated with closure of the two atrioventricular valves and the second sound with closure of the two arterial valves. Current thought has been along the line that tensing of the valve curtains, not collision of the margins of the cusps, is the basis for the sound produced. Dock, like Rouanet in the early 1830's, has done experiments tensing valve segments with the production of noises like heart sounds. Dr. Francis Palfrey, in teaching physical diagnosis at Harvard Medical School, was in the habit of tensing his pocket handkerchief to produce sounds like the heart sounds, with a greater length of handkerchief for the first sound than for the second (1176).

Since *in vivo* the valve curtains are loaded by blood, experimental tensing of fresh pericardium or valve segments in air is not completely analogous. This fact was recognized by Dock (307), who had his experiments arranged so that the tensed strips were submerged in water.

It is probably most accurate to think of sounds such as the first and second sound and the mitral opening snap as hydrostatic pressure transients produced by the abrupt interruption of the momentum of a local flow (Fig. 85) (e.g., before closure of the atrioventricular valves there must be slight local flow accompanying the billowing of the valve curtains toward the atrium). The valve curtains and the chordae which support them are inextensible collagenous structures. When their limit has been reached they interrupt the local flow abruptly with a translation of energy into a pressure transient.

In the case of the closure of the arterial valves resulting in the second heart sound there similarly appears to be a local backflow toward the bellies of the valve cusps. The interruption of this backflow again incites a pressure transient which, after transmission to the surface of the chest and detection by appropriate means, is designated "the second heart sound."

In connection with the point of view just explained it should be noted that the pressure transients are located predominantly in one chamber or vessel. The first sound in the ventricles, the second sound in the base of the aorta or pulmonary artery, the mitral opening snap (see below) in the left atrium, etc. This fact accounts for the findings of intracardiac phonocardiography, particularly the pronounced attenuation from one chamber to the next and the fact that S_1 is loudest in the right ventricle, whereas the second sound is loudest in the pulmonary artery.

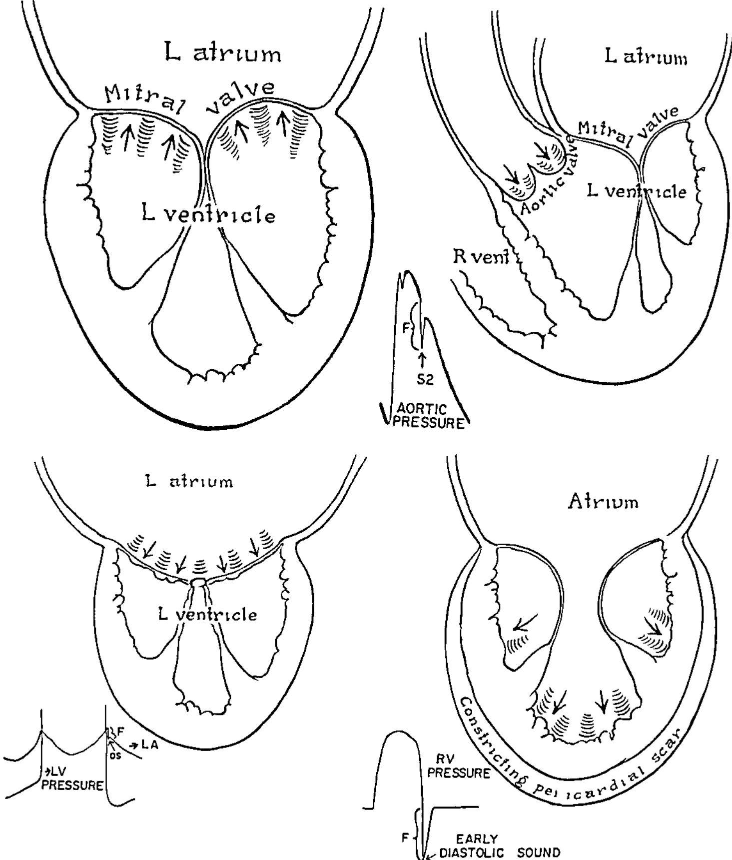


Fig 85 A schematic representation of the inciting of pressure transients when a valve in closing brings an abrupt halt to local flows (*Left top*) Mitral (or tricuspid) closure The aortic valve has not yet opened Note that the pressure transients are conceived as being created mainly in the ventricle The resulting sound is heard mainly at the apex (*Right top*) Aortic (or pulmonary) closure The pressure transients are conceived as being created mainly in the great vessel The resulting sound is heard mainly at the base of the heart (*Left bottom*) Mitral valve opening in mitral stenosis The pressure transients are conceived as being created mainly in the left atrium The resulting sound is heard mainly at the left sternal border but also at the aortic area and in the suprasternal notch (*Right bottom*) The early diastolic sound in constrictive pericarditis Pressure transients are generated when ventricular flow is brought to an abrupt halt by the constricted ventricular wall after the capacity of the ventricle has been attained In pressure curves (inserts) F = time of local flow arrow = time of sound

(896) Furthermore it accounts for some of the topography of the valve sounds on the surface of the chest. Is it any wonder, for example, that the first sound is loudest at the apex and the second sound at the base?

The above point of view is stated by Rushmer (1323) as follows:

Since the chambers of the heart are filled with blood, none of these structures can vibrate independently without producing movement of the blood. Similarly, vibrations in the blood must be transmitted to the surrounding structures. If the sounds can be picked up from the external surface of the body, all structures between the heart and the thoracic wall must be vibrating. It is futile to consider vibrations of the heart wall, valves, arterial wall, and blood individually when in fact they constitute an interdependent system and all vibrate at the same time. A more realistic approach to the problem results from considering those conditions which lead to vibrations of cardiohemic system composed of the blood, heart walls and valves.

Rushmer suggests that the heart sounds are the result of abrupt acceleration or deceleration. The valve closure sound, the mitral *opening* snap and the sound of constrictive *pericarditis* are seemingly the product of abrupt *deceleration* of local flow. Whether abrupt *acceleration*, as with valve opening, can produce sound is less convincingly demonstrated but clinical experience would suggest it can. The sound generating properties of opening valves should be studied in models simulating the heart.

Kassatkine (709) also seems to have conceived of the same general mechanism outlined above in his analogy *to the hydraulic ram*, although ingenious, may not be entirely applicable.

The students of conventional oscillographic phonocardiography have divided the heart sounds into several components, to each of which a separate origin has been assigned. The likelihood of fallacy in assigning separate significance to individual squiggles in an oscillogram is *a priori* enormous. Furthermore, *if one* divides the heart sounds into four components, as has been fashionable, then one should divide them into eight inasmuch as *there* are really two simultaneously functioning *heart*s, each capable of producing the four types of noises (862).

In the case of the first heart sound components have been ascribed to the contraction in the myocardium itself and some to opening of the arterial valves and/or to movement of blood out of the ventricle. At the onset of the first sound there are vibrations which have been thought to be the residual effect of atrial contraction. This complicated schema has little evidence to support it. Although it is long established that the contraction of skeletal muscle (399, 677) and presumably of myocardium is productive of a sound, the sound must be of low frequency and intensity.

Most recently Luisada (982) has championed the existence of four main components in the first sound and relates these to mitral closure, tricuspid closure, aortic opening and pulmonic opening. Usually he thinks AV valve *opening* is noiseless because it is occurring at low pressures. On the other hand he thinks that the opening of *the arterial* valves at high pressures causes a shaking which is expressed as a sound. Intracardiac phonocardiography and animal experiments provide support for the above view. On the other hand Reinhold and Rudhe (1257) with electrokymographic correlations could find support for the existence of mitral and tricuspid components (*in that order*) in the first sound but none for a contribution by arterial valve opening. They, like Luisada, concluded *that normally* AV valve opening is noiseless.

There is a low *frequency* component preceding the sound(s) of *closure of the* AV valves (1139). This was interpreted as representing "residual atrial vibrations" by Cossio and Lascalea (301), by Orias *and Braun* Menendez (1166) and by Rappaport and Sprague (1244). *Later the* observation that *these* vibrations persist in the presence *of atrial fibrillation* (708) made this explanation untenable. They occur after the onset of the QRS *of the* electrocardiogram (908) and although this fact does not exclude atrial origin, they can therefore be related usually to myocardial contraction preceding closure of the AV valves.[1] The

[1] Vibrations preceding the major component of the first sound in cases of mitral stenosis probably have a different explanation (see p. 287). In brief, these are thought to represent the *tricuspid* closure sound rendered audible by the paradoxical sequence of *mitral and* tricuspid closure in mitral stenosis. The possibility exists that some of the *recordings* on the basis of which it was concluded that the *initial* vibrations persist in atrial fibrillation were made in *patients* with mitral stenosis and that in fact the vibrations in question represented tricuspid closure sound.

low frequency composition of this component is consistent with a muscular origin[2] Since the right ventricle usually begins to contract first but the mitral valve closes first, the interval between the onset of the "initial vibrations" and the "fast vibrations" of the first sound cannot be used as a measure of Kelly's (779) mechanical lag phase

The occasional atrial origin of the initial vibrations cannot be completely excluded Duchosal (377) found that as a patient with atrial gallop improves clinically the interval between the gallop and S_1 becomes less and at times the atrial gallop becomes part of S_1 Leonard and his colleagues (876) found that in cases of atrial gallop in hypertensive patients application of tourniquets to the extremities causes the atrial sound to move progressively into the first sound and assume the appearance of the initial vibrations This suggests that the initial vibrations may sometimes be atrial in origin See also Lian's "galop presystolique retardé" (p 175)

In early systole there is frequently a very short ejection murmur even in normal persons which in the oscillographic phonocardiogram, in particular may be interpreted as a prolongation of the first heart sound When aortic stenosis or pulmonary stenosis is present a forward snapping of the stenotic valve membrane or an abrupt cessation in the opening process may produce a snap intimately related to the first sound

When dilatation of pulmonary artery or aorta occurs, especially if hypertension is also present in the great vessel in question, a click in early systole may occur and may be interpreted as part of the first sound The *early systolic click* may result from a snapping of the vessel wall with ventricular ejection or, more likely following the observations of Hultgren (725) from a snapping of the inextensible valve ring or annulus fibrosus Hultgren could record no snap directly over the great vessels but could record a snap over the annulus fibrosus The most effective way to produce the sound artificially was to place a band of inextensible material around the main pulmonary artery or aorta, without producing compression

It should be pointed out that some believe the early systolic click is an exaggerated form of the normal opening sound of the aortic and/or pulmonary valves They state that this exaggeration occurs not only with disease of one of the two great arteries but also with either aortic stenosis or pulmonary stenosis Davidsen (350A) goes so far as to suggest a different terminology—"pulmonary first sound" (1^P sound)—for the now current "pulmonary early systolic click"

The students of oscillographic phonocardiography have divided the *second heart sound* also into four components three in addition to the noise of closure of the arterial valves initial vibrations of ventricular relaxation, a sound of opening of the AV valves, the after vibrations in the great vessels above the valves The only possibility worthy of serious consideration is that of a normal opening sound arising at the AV valves In mitral stenosis and tricuspid stenosis such occurs quite as a rule as the so called "opening snap" In conditions of high flow across the AV valves the question of an opening snap without actual stenosis has been raised for example Leatham (863) has made reference to a tricuspid opening snap in atrial septal defect Although perhaps a faint AV opening sound will be found on intracardiac phonocardiography in normal *it is doubtful that such sound can be detected on* the surface of the chest (p 84) Low frequency vibrations which have been seen at the end of the second sound in oscillograms and ascribed to AV valve opening (983) would seem to occur too close to the second sound to be this judging by the experience with the bona fide opening sound of mitral stenosis

In connection with the production of the *physiologic third heart sound* and the *protodiastolic gallop* which is generally conceded to represent a pathologic exaggeration of the former, two main suggestions[3] are promulgated (1) that they repre

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sent vibrations set up in the ventricular myocardium by rapid ventricular filling and (2) that they represent a re-closure sound of the AV valve or at least that a reflux with rapid ventricular filling occasions tension of the curtain and/or the chordae of the AV valve with production of the observed sound. The critical experiment has not yet been devised and there are bits of evidence suggesting or contradicting both suggestions. For example, in kymographic studies of ventricular border motion in patients with a protodiastolic gallop Brady and Taubman (149) described an anomalous medial motion at the time of the gallop sound. They presented this observation as evidence of the reflux necessary to the second theory. An extracardiac contribution to these sounds was suggested by Dunn (382) who reasoned by analogy to the early diastolic sound of constrictive pericarditis, and by Harvey (602) who in describing what he termed "ventricular knock" and what is almost certainly merely an exaggerated protodiastolic gallop occurring in association with mitral regurgitation observed a striking impact of the heart against the anterior chest wall.

Also at various times the view has been expressed that the physiologic third sound is a physiologic opening snap of the AV valves. As recently as 1954 (402) a study which appeared to support this notion consisted of correlation of the jugular venous pulse with the heart sounds. Lag in the former (828) probably accounts for the seeming correlation between the peak of the V wave and the third sound.

The idea that the third heart sound arises largely in the ventricular myocardium is attractive. It would not seem inconsistent to think that tautening of the chordae and/or cusps of the AV valves also occurs as the ventricle elongates and contributes to the third heart sound. The variation in quality of the third heart sound—from a dull thudding sound of low frequency in the SLCC to a sharp even snappy sound with correspondingly different pattern in the SLCC—is consistent with a dual mechanism and varying proportions of the two contributing factors. Orias (1105) also points out that in locating the origin of the vibrations in the ventricular wall one is not excluding tendinous and valvular structures from participation in the tensing process.

One bit of evidence which is usually overlooked in discussions of the genesis of the third heart sound and which is against the purely valvular origin of the sound—at least against re-closure as the mechanism—is provided by Rytand's demonstration (1336) that in atrial fibrillation S_3 is loudest when it occurs in that part of the cardiac cycle in which the normal third heart sound occurs (see Fig. 447, p. 437). This would suggest that the AV valves are flung most widely open at this time. To be sure, an alternative explanation agreeable to the "valvular school" is that there is a summation of valve closure sounds—the ordinary third heart sound and the first heart sound—to account for the observations noted.

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Regardless of the details of mechanism two factors seem to be involved in the production of the protodiastolic gallop: (1) rapid ventricular filling and (2) alteration in the volume elasticity characteristics of the ventricle.

There is some difference of opinion as to whether a protodiastolic gallop can be produced in the right ventricle. In the experience of Wolferth and Margolies (1577) three of 60 gallops were right sided. On the other hand Frost (498) seems to have thought that all gallops are produced in the left ventricle. The occurrence of gallops with cor pulmonale and with pulmonary stenosis would seem adequate evidence that the right ventricle can produce gallop sounds.

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of correlated recordings (see Fig. 42b), be demonstrated to be related to abrupt halt in ventricular filling. It is a "water hammer" sound or "cocktail shaker" sound. Most students now hold that this sound is fundamentally the same as the protodiastolic gallop and that its unusually close position to the second heart sound is merely a function of the unusually high venous pressures—and *therefore unusually rapid ventricular filling*—which are features of constrictive pericarditis. However, almost never in congestive heart failure on other bases does one encounter a protodiastolic gallop quite as close to the second sound even with venous pressures in a comparable range. *Therefore, the limitation on diastolic filling* with an abrupt halt in filling must be a factor in the timing of the sound. The protodiastolic gallop of pericardial effusion probably has a similar basis—impact vibrations are produced when the capacity of the ventricle is attained. The protodiastolic sound of constrictive pericarditis illustrates the production of a sound from abrupt deceleration (see Fig. 85D).

The atrial or *fourth heart sound* occurs normally in children and occurs in exaggerated form in adults as a presystolic gallop or in association with heart block. In connection with this sound there are protagonists for a myocardial origin and those for a valvular origin, as in the case of the protodiastolic sound.

Oscillographic studies reveal two separate components to the atrial sound, each probably with its own mechanism. The first element is always audible in the esophagus and probably is related to tensing of the contracting atrial wall. That the sound indeed has this basis may be supported by the facts that an atrial sound may be found in the midst of ventricular systole in cases with nodal rhythm and retrograde atrial excitation (376) and in cases with complete atrioventricular dissociation (346). The second element is recorded mainly from the precordium and probably is a sound generated in the ventricular myocardium or possibly the AV valve mechanism as a secondary result of the contraction of the atrium.

The unusually snappy character of the atrial sound in many cases of presystolic gallop may indicate that tensing of the AV valves is involved. Henderson and Johnson (670) in the now classical paper in which the role of atrial systole in closure of the AV valve was defended, went much further than more recent evidence would permit and insisted that closure of the AV valves occurs normally in presystole and that only with atrial fibrillation and other situations in which atrial contraction is missing, at least from the usual position in the heart cycle, is there what they thought of as a "hinge mechanism" of closure of the AV valve through elevation of ventricular pressure. This idea is still consistent with the view that the first heart sound (which occurs after the onset of ventricular contraction) is due to tensing of the AV valve, since this tensing could occur even in a *closed valve*. However, the absence of an audible atrial sound except in a small number of young subjects—it can be demonstrated in recordings from the esophagus in all persons—seems against a complete closure of the AV valves with atrial systole under normal circumstances. The possibility *certainly remains* that such occurs with unusually strong atrial contraction, that is, in the conditions in which a presystolic gallop occurs.

Leonard and co-workers (876) believe that the presystolic gallop of arterial hypertension occurs earlier than the normal atrial sound of which it is a pathologic exaggeration. The normal atrial sound may occur so late as to fall after the onset of the QRS. What is interpreted as "muscular component" of S_1 may be in fact an atrial sound in some instances (see p. 126). By cutting down on venous return to the heart by venous pooling, Leonard *et al.* (876) could demonstrate that the presystolic gallop will decrease in intensity and move progressively toward or even into the first sound. Seemingly venous pooling has normalized the situation with respect to both hemodynamics and heart sounds. The audibility of the exaggerated and abnormally early atrial sound in systemic arterial hypertension is *enhanced by delay in the first heart sound* (p. 427).

In discussions of the mechanism of gallop sounds it is likely that a careful distinction should be made between the two types. We do not know that the mechanism of the two is identical. Furthermore, there is some reason to suspect it is different since the clinical setting for the two is in a general way different (see p. 175).

Clicks occurring *in systole* are believed to arise

in extracardiac structures for the most part. The early systolic click of dilated aorta and pulmonary artery, particularly with hypertension in the vessel in question, appears to arise through a snapping or tensing of the vessel wall early in ejection. Hultgren (725) presents evidence in support of the view that it is snapping of the inextensible valve ring which is responsible for the sound. Leatham and Vogelpoel (865) suggested an identity to the "ejection vibrations" recorded by Wiggers and Dean (1554) on the exposed great vessels of the dog. Finally, Luisada and colleagues (982) insist that the early systolic click is an exaggerated form of the sound which they think normally occurs with opening of the arterial valves and constitutes the latter part of S_1. What they apparently conceive of are vibrations incited when the ejected blood first meets the static blood in the base of the aorta.

Mid and late systolic clicks are commonly produced by the tensing of pleuropericardial adhesions. Supporting this statement, the evidence, such as it is, is as follows: (1) These clicks are often observed to appear following an attack of acute pericarditis. (2) They may occur in patients with extensive pleural and pulmonary disease by tuberculosis (Fig. 141), sarcoid (76A), etc. (3) Pleuropericardial adhesions have been observed at necropsy in patients who manifested clicks in life. (4) Luisada (981) observed on fluoroscopy a tugging at one leaf of the diaphragm synchronous with the systolic click. It is difficult to understand why the click or clicks should be late in systole when this is the mechanism. Sometimes the systolic click initiates a systolic murmur which is usually noisy but sometimes musical and which is believed to result from the rubbing together of roughened pericardial surfaces. Although the early systolic click is most often produced in the aorta or pulmonary artery, pleuropericardial adhesions at times produce an early systolic click. Furthermore, a click with this mechanism may occur in early diastole (76A). Usually, however, there are in such cases other click(s) in late systole.

Although the above arguments for pleuropericardial adhesions as the basis for systolic clicks has failed to convince some (604A) (1579A), most would agree that they are of extracardiac source. They bear no constant relationship to one part of the cardiac cycle. Their position varies from early systole to early diastole and is influenced by respiratory phase and position of the subject.

Sometimes mid systolic clicks appear to be due to movement of costochondral or chondrosternal joints by an enlarged heart, or by a normal heart which is in abnormally intimate contact with the bony cage of the thorax because of pectus excavatum or other deformity of the chest or of displacement by ascites, pregnancy or intrathoracic mass. Loose jointedness as in the Marfan syndrome may contribute to the production of these sounds.

It is debatable whether there is any category of transient located between the first and second sound other than the clicks produced through the mechanisms mentioned above. Specifically, is there anything which can suitably be called a systolic gallop? Occasionally we have observed in cases of aortic regurgitation a mid systolic transient of lower frequency content, slightly greater duration and in general more thudding quality than the usual systolic clicks. The mechanism is obscure. It may be produced in the aorta when full capacity is reached. The large stroke volume of aortic regurgitation may account for its occurrence in association with this valve lesion.

There remains to discuss one more category of transient, namely, the *opening snap of the AV valves*. Luisada has thought that small vibrations recorded at the surface of the chest can at times be related to opening of these valves in normal subjects and that in intracardiac phonocardiograms such vibrations can be frequently identified. Opening of the mitral or tricuspid valve may be associated with a striking snap when stenosis of the valve is present. The adhesion of the commissures in these cases results in a fibrotic diaphragm which billows or, better, "snaps" toward the ventricle or toward the atrium when the polarity of the atrioventricular pressure differential is reversed (Fig. 86). Abrupt bellying of the membrane toward the atrium (as well as tensing of the shortened, fibrotic chordae tendineae) contributes to the snappy first heart sound of mitral stenosis. Ballooning in the opposite direction in early diastole produces the mitral opening snap

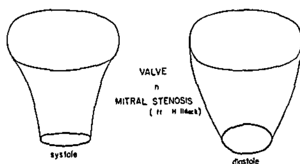


FIG 86 Mechanism of opening snap

The abrupt billowing of the mitral curtain toward the atrium early in ventricular systole and toward the ventricle in early ventricular diastole is a possible explanation for the snapping first sound and the mitral opening snap respectively in mitral stenosis (Adapted from Holldack (701))

Theoretically the opening snap might be expected to be more intense in the left atrium than in the left ventricle The pressure wave created when *the inextensible aortic leaflet of the mitral valve* brings local flow abruptly to a halt is located in the left atrium The characteristic location of maximum audibility of the mitral opening snap on the surface of the chest is consistent with the theoretical consideration If it were not for the fact that the pressure transient produced by closure of the mitral valve is located predominantly in the left ventricle and the pressure transient produced by opening of the mitral valve located predominantly in the left atrium, it would be difficult to explain the differences in location of *maximum audibility of* S_1 *and the opening snap* —sounds generated at the same structure An analogy has been drawn to a child's toy cricket" in which a metal tongue which is stiff, yet flexible produces a snap when pressed with the thumb The analogy is *not appropriate since the anterior* (aortic) leaflet of the mitral valve does not have stiffness comparable to that of the tongue of the "cricket " Bending of the metal is rather directly responsible for the sound in the cricket whereas in the opening snap the primary event is the pressure transient(s) created in the fluid when there is abrupt deceleration of local flow by the inextensible valve curtain (See Fig 85C)

Snappy sounds occurring shortly after the second heart sound and possibly *representing opening snaps* of normal valves occur in instances of very large diastolic flow across the valve such as the tricuspid valve with atrial septal defect or the mitral valve with ventricular septal defect and *patent ductus arteriosus* This is, however, as yet an inconsistent and far from established phenomenon It is somewhat clearer that an opening snap can be produced in an AV valve which is not stenotic but which is scarred as a result of rheumatism or of fetal fibroelastosis

MURMURS

Most murmurs are noises, the genesis of these will be discussed first Other murmurs are musical, the special features of the genesis of these will be discussed later

The ideas that will be developed in the following discussion are as follows

1 Turbulence and murmur cannot be equated to each other in any direct manner The Reynolds number provides information only of qualitative and descriptive nature and is largely of conceptual usefulness The Reynolds formula is useful largely as a catalogue of factors involved in the genesis of murmurs

2 Most murmurs arise through a complex interplay of disturbed flow and the wall and other boundary structures

3 *Cavitation* should be considered as a possible mechanism in murmur production

After a survey of basic physical principles pertinent to murmur production, the classic concepts of the generation of murmurs will be reviewed

Thrills and murmurs are basically the same phenomenon sensed in a different manner Therefore what is said about the genesis of murmurs applies to thrills also One frequently hears murmurs without being able to feel a thrill, but whenever *there is a true thrill felt there is* also a murmur, usually a loud murmur The frequency response curves of the fingers for palpation of thrills and of the ear for the hearing of murmurs are different palpation is most efficient in the *low frequency range, audition in* the higher frequency range

TURBULENCE The reader is referred to pages 47 and 48 for description of the experiments on the basis of which Reynolds defined the factors *responsible for transition from* laminar (streamline) to turbulent flow The Reynolds formula is as follows

Reynolds number (R N) =

$$\frac{\text{diameter of conduit} \times \text{velocity of flow}}{\text{kinematic viscosity of fluid}}$$

(kinematic viscosity is absolute viscosity divided by density) The Reynolds number is dimensionless Furthermore *it represents a law of similarities* Providing that the conditions of the basic experimental model on which it is based obtain the transition from laminar to turbulent flow will, for example occur at a Reynolds number of about 2000 for a wide range of flow velocities varieties of fluid as fr as viscosity and density are concerned and sizes of tubes

The difficulty encountered in attempting quantitative application of the Reynolds formula to the circulation is that the conditions of the basic model do *not* obtain The interior of the heart and base of the great vessels in the vicinity of the valves—sites of greatest interest from the stand point of murmur production—are obviously quite different from the model The aorta too is fr from a long straight tube of uniform diameter (Fig 87)

However there is a sizeable body of evidence that the Reynolds formula is of some pred tive value as to the influence that changes in the Reynolds parameters will have on the intensity

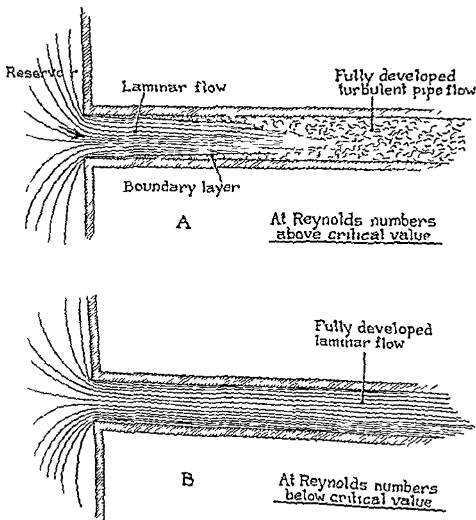


Fig. [illegible] The drawings illustrate the "entrance length" before the flow pattern—turbulent or laminar—is establ hed In the case of laminar flow the drawing attempts to represent the parabolic velocity profile with most rapid flow at the center of the pipe

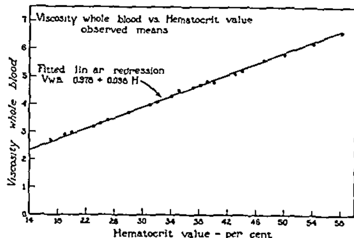


Fig 88 A Relation of blood viscosity to hematocrit (From Nygaard Wilder and Berkson (1149))

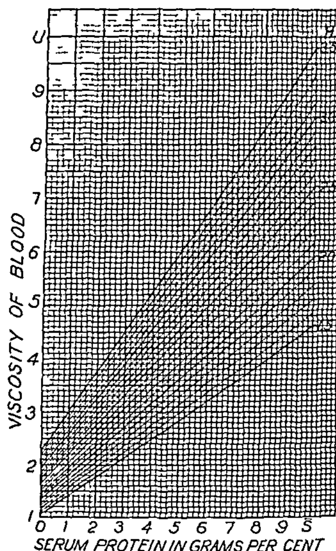


Fig 88 B The relation of blood viscosity to the concentration of plasma proteins (From Lamport (836))

of existing murmurs or the occurrence of murmurs not normally present

1 Reduction in kinematic viscosity in anemia (Fig 88A) is attended by the development of murmurs

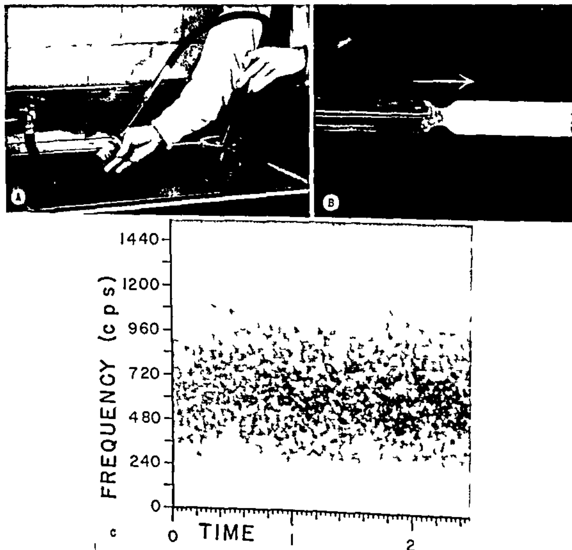
2 Increase in kinematic viscosity in polycythemia or hyperproteinemia (Fig 88B) suppresses murmurs which would be present with normoerythemia and normal levels of serum proteins

3 Localized dilatations in vessels, such as in aneurysms of the aorta, are the site of murmur production *Dilatation of the ventricle beyond an* AV valve ring of normal dimensions is a comparable situation

4 Increased velocity of flow—and increased volume of flow through a conduit of normal caliber which amounts to the same as increased velocity of flow—is accompanied by the production of murmur in thyrotoxicosis with exercise or injection of epinephrine This factor is probably also operating in the case of anemia The increased volume of flow across a normal AV orifice—the tricuspid orifice in atrial septal defect the mitral orifice in ventricular septal defect and patent ductus arteriosus—is likely to be accompanied by murmur (Dilatation of the ventricle probably contributes in these instances) Increased stroke volume in aortic regurgitation often produces a systolic murmur in the aortic area Increased flow across the mitral valve in mitral regurgitation may produce a diastolic murmur or exaggerate the murmur of any mild mitral stenosis which may be present

The venous hum (see p 226) illustrates very clearly the application of Reynolds critical velocity concept to the generation of murmurs Flow in the veins basically streamline (666 667) may become turbulent with appropriate change in any of the factors in the Reynolds formula and murmur develops

That turbulence is not the whole story in murmur production and that turbulence and murmur cannot be equated to each other directly is suggested by the fact that in tubes with rigid walls Reynolds numbers far in excess of the critical or transitional value are attended by little or no murmur (879) However a murmur does occur when the same flow occurs in a tube with walls of a flexible material such as rubber or plastic For a murmur of appreciable intensity to be produced the boundary structures must be flexible



Fic 83 When water i cau e l to flow through a tube with a constriction (A) if the velocity of flow is sufficiently high bubble will form at the neck (B) and produce a hi ing noi e of which the soun l pectrogram is shown in (C) The level of frequency di played by the cavitation noi e generate l in this rigid walle l tube is in the upper range of that een in som murmurs

enough to vibrate in response to di turbed flow in the lumen of the vessel or heart [4]

McDonald (1060) uggests that vortices or eddies hould be di tingui hed from turbulence and that the former may be repre entative of the character of the flow di turbance at diseased valve orifices as ociated with murmurs

CAVITATION [5] The reader is referred to pages 47 and 48 for a di cus ion of the historical back ground of Reynold cavitation Reynolds appears

[4] In 1956 Laul rs and Pe (84) came to a imilar conclu ion They in isted that Savart s fluid vein (p 45) wa ina lequate to account for murmurs and that el oes succe ifs of the fluid veins on boundary struc ture are re p n il le

[5] The experience and experiments relating to the form of cavitation re pon ible for the 1 ends (Caisson s di ea e decompre ion sickness) are pertinent in con nection with cardiovascular sound (5 1063) but cannot answer the direct que tion of whether cavitation indeed plays a role

to have been more impressed with this as a mechanism for sound generation in flowing liquid than with turbulence. The structural similarity of the Reynolds tube (Fig. 89) to valve stenoses and coarctation of the aorta raises the possibility that the drop in pressure at the constriction in accordance with the principle of Bernoulli may result in bubble formation which in turn is accompanied by murmur production.

The following armchair thoughts might indicate that cavitation is an unlikely basis of murmurs.

1. In the cardiovascular system there might never be sufficient velocity of flow at constriction to produce the drop in pressure necessary for bubbling.

2. If the necessary drop in pressure does occur, might it not result in collapse at the neck of the *constriction before bubbles would develop?* A musical type of murmur by a "flutter" mechanism might be anticipated more than cavitation. (How*ever, "flutter" and "cavitation" are not mutually* exclusive phenomena.)

3. Cavitation which occurs in association with *improperly engineered ship propeller blades* is a violently corrosive and destructive phenomenon. Might not intolerable damage be produced in the walls of the cardiovascular system and the valves? Cavitation damage is avoided by the use of a rubber coating of propellers. The softness of the walls of the cardiovascular system may have a similar protective effect.

4. The large and abrupt change in pressure at the neck of the constriction and the hammering effect of the bubbles might be expected to produce disruption of the erythrocytes.

The bubbles which form in the process of cavitation may consist either of dissolved gases or of the vapor phase of the liquid in question—in the case of blood, water. One might think that favoring the possibility of cavitation is the fact that the blood has relatively high partial pressure of gases. In fact, cavitation in the form of water vapor appears to occur with greater facility than cavitation from dissolved gases.

Even if Reynolds cavitation does not occur in the circulation, and such indeed seems unlikely, it is possible that cavitation of some special variety does. For example, the pressure drop in the center of an eddy, the importance of which is emphasized by McDonald (1060), might be adequate to initiate bubble formation.

Certain *classical mechanisms* for murmur production can now be reviewed (Fig. 90).

1. The abrupt widening of the conduit or the resumption of normal caliber beyond a constriction is productive of a murmur. The mechanism here is undoubtedly complex. Turbulence and/or eddy formation in the fluid and, possibly, cavitation are important; the disturbance of flow which these represent causes the wall to vibrate. There may be direct *driving of the wall just distal to* the constriction through the opposing forces of the wall elasticity and the centripetal force described by *the principle of Bernoulli*.

2. Free flow at high rates can be accompanied by the production of murmurs. The interplay of *turbulence with the vibrating wall is probably* the basic mechanism.

3. Jet impact—direct driving of a wall opposite *an orifice at which the jet is produced*—is probably an important mechanism in patent ductus arteriosus, peripheral arteriovenous fistula, coarctation, aortic stenosis, and *possibly mitral re*gurgitation. In some of these situations jet lesions—a localized patch of atherosclerosis—develop *at the site of impact of the jet (409).*

Jet lesions[6] occur in many parts of the cardiovasculature when because of acquired or congenital abnormality of architecture there is a small orifice with a high pressure differential on its two sides. Jet lesions are useful anatomic labels for sites of murmur production. *In addition to patches* of atherosclerosis at the point of traumatization by direct impact, there may be cusp-like pocket-*like lesions, sometimes referred to in the older* literature as Zahn's or Schmincke's pockets. These appear to represent a scuffing up of the endocardium *by the regurgitant stream impinging* somewhat obliquely on the surface in question. In bacterial endocarditis organisms may lodge on *jet lesions*.

In *aortic regurgitation,* the jet may strike the interventricular septum (most frequently), the *aortic leaflet of the mitral valve* (with production of an Austin Flint murmur) or the anatomical apex of the left ventricle (with production of

[6] I am indebted to Dr. J. E. Edwards, Rochester, Minn., for many details of this section.

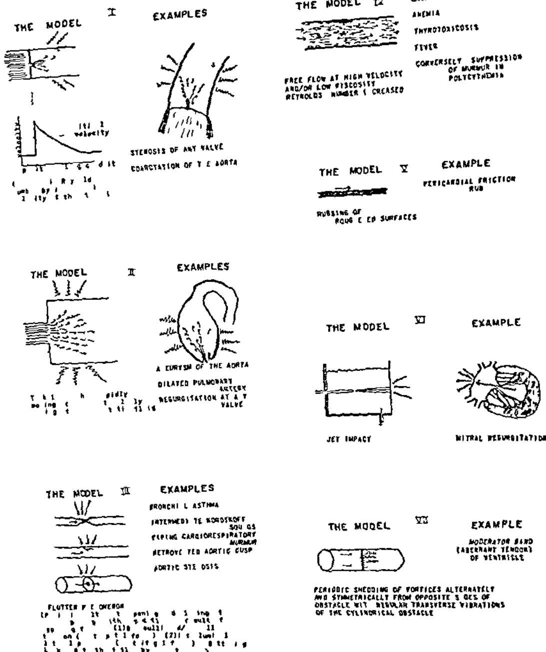


Fig 90

predominant axillary location of the murmur—the Cole Cecil murmur) With aortic regurgitation without steno i a jet lesion may develop in the a cending aort i as a re ult of the re-orientation of the left ventricular outflow tract to the aorta (because of left ventricular enlargement) and as a result of the large stroke volume The occurrence of a loud sy tolic murmur accompanied by sy tolic thrill in cases of syphilitic aortic regurgitation may be related to this jet le ion

In *mitral regurgitation* the most common site of a jet le ion is on the posterior wall just above the

posterior cusp This fact correlates well with the well known axillary "radiation" of the murmur of mitral regurgitation It is likely that the so called MacCallum's patch (1004) is in many cases a jet lesion rather than a lesion resulting directly from rheumatic fever (174, 408) Edwards and Burchell (412) describe one case in which because of the particular location of the valve lesion in bacterial endocarditis, the jet impinged on the inter atrial septum In this case a systolic murmur and thrill was incorrectly interpreted as indicating aortic stenosis I have seen cases which are probably identical The murmur and thrill were maximal in the *third* right interspace

In pure *pulmonary stenosis* there is likely to be a jet lesion in the region of the bifurcation In tetralogy of Fallot with infundibular stenosis the jet lesions may be in the infundibular chamber and on the ventricular side of the pulmonary valve

In *tricuspid regurgitation* jet lesions are rare, probably because of a relatively low pressure in the right ventricle but have been described in primary pulmonary hypertension (408)

It is the small *ventricular septal defect,* with normal right ventricular pressure and high pressure differential across the defect, that is likely to show jet lesions In brief it is the classical *maladie de Roger* which has such lesions Since the left ventricle is posteriorly located whereas the right ventricle is the anterior ventricle the jet is directed in the postero anterior direction The locale of the murmur and thrill in Roger's disease corresponds to the position of the jet lesion in the right ventricle

In *atrial septal defect* pressures are usually not sufficient to produce jet lesions

In *patent ductus arteriosus* jet lesions occur in the left pulmonary artery With the artificial ductus created by the Blalock Taussig operation the jet lesion is usually located farther peripherally in the pulmonary artery because of the orientation of the subclavian artery in relation to the pulmonary artery

Edwards has pictured (414) jet lesions of the intima of the aorta distal to a coarctation

4 Pericardial friction rubs are murmurs too The mechanism is one of "stick and slip" In the usual variety of pericardial friction rub, "stick and slip" at innumerable points in time and in location over the pericardium are *responsible for* the sound Sometimes by ear and even more clearly by SPCG pericardial friction rubs are seen to be made up of a series of closely spaced clicks This suggests that in these instances there is a finite number of "stick and slip" events within, at least, a certain area surrounding the point of auscultation

Musical murmurs differ from noisy murmurs in two striking respects which are probably highly significant with reference to genesis (1) Considered as a class musical murmurs are more intense than noisy murmurs Most murmurs audible at a distance from the body are largely musical in character A colleague of mine heard a

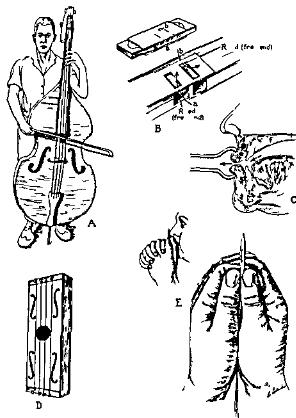


FIG 91 1 Counterparts in musical instruments of generators of musical murmurs

1 Bass viol (violin family in general) analogous to musical pleuropericardial murmurs *B* Harmonica not strictly analogous to retroverted aortic cusp since the reed faces into the wind—not downstream Reed *a* is activated on blowing and alternate reed *b* on sucking *C* Trumpeter's lip analogous to calcific aortic stenosis *D* Aeolian harp analogous to anomalous chordae tendinae *E* Reed of grass More like a downstream reed but differs in that the fluid stream passes on both sides of the vibrating member

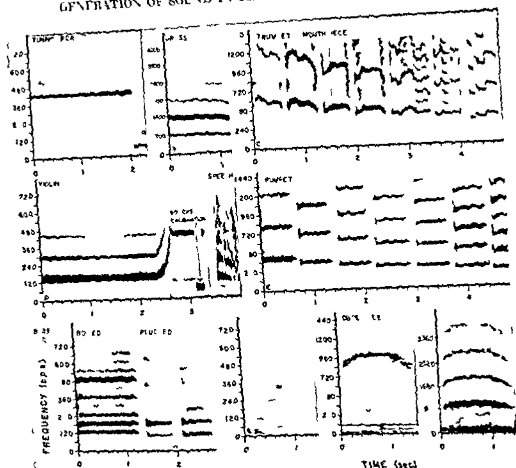


Fig. 91B Spectrograms of musical sounds

A Tone of tuning fork 440 c p s and on right 60 c p s calibration. *B* Musical sound produced by blowing on a reed of grass. Note that the second harmonic is louder than the first which presumably is the fundamental. This was also the situation in the case of the musical tone produced by a silk thread across a water conducting tube (1083) (See Fig 94 A). *C* Trumpet mouthpiece. The tones are not steady and impurities in the form of additional partials are present in certain notes. *D* Violin with portamento (glide) from about 150 c p s up to about 440 c p s. A brief segment of speech sound is also present and a 60 cycle calibration. *E* Full trumpet. Here the tones are steady and pure. *F* Musical sounds of a bass viol. *G* Recording of musical sound produced by blowing on isolated oboe reed. These are analyses of the same sound on three different frequency scales. Below 720 c p s there is nothing except wind noise. The fundamental located at 960 cycles appears in the analysis to 1440 c p s. A total of three overtones are demonstrated in the third analysis.

"sea gull" murmur in church. The spouse of the patient may complain of the noise in bed (182). (2) Post mortem one finds in association with each variety of musical murmur some elastic boundary structure—stenotic diaphragm, valve flap, pericardium, wall at the tendinous cord-like structure—which is set into vibration in a specifically periodic and therefore musical manner. Musical tones can be produced by the flow of fluid in a perfectly rigid system. For example the shedding of vortices off the sides of a rigid cylindrical obstruction, the so-called Kármán streets, may produce a musical tone and obviously the flow of air in an organ pipe is productive of a musical tone without the presence of an elastic flap which is free to vibrate. However, no such rigid structures occur in appropriately intimate relation to pertinent portions of the cardiovascular system.

In the generation of musical murmurs it is useful to compare the modus operandi of the generator to that of various musical instruments (Fig. 91 A and B). The physics of musical instruments has been extensively studied and much

of the information so gathered is applicable to musical murmurs

According to the type of musical instrument most analogous, four general groups of musical *instruments are clearly identified, plus a fifth* less easily analogized group

TRUMPET GROUP It is possible to play a respectable scale with only the mouthpiece of the trumpet The function of the rest of the instrument is to introduce purity and control The anatomical situation in the case of calcific aortic stenosis is analogous The stenotic valvular diaphragm is analogous to the trumpeter's lip The pitch of the note produced is determined by the stiffness of the lips, the degree of their separation and the velocity of the fluid stream Whistling is another analogous situation Vibration of the lips in whistling can be demonstrated by holding the lips, which stops the whistle

AEOLIAN HARP GROUP Musical murmurs generated at aberrant chordae tendineae, or aberrant tendons ("moderator bands") of the ventricle are analogous to the musical tones produced by the Aeolian harps which in the past were placed in the windows of villas or castles so that the breeze, in blowing through the wires would produce a bit of music In those cases in which aberrant chordae traverse an interventricular septal defect, the analogy is striking indeed In association with Chiari's network of the right atrium a musical murmur which is continuous may be produced (see p 410) Yet another example may be the musical murmur which develops at times over the ascending aorta in cases of dissecting aneurysm and which may be produced by flow past the fibrous cords which traverse the false channel The murmurs in this group are what Bondi (129) referred to as *Fadengeräuschen*

REED GROUP *In the case of retroverted aortic* cusp, the anomalous member seems to function like the reed of a musical instrument However whereas the retroverted cusp points downstream since it is activated during ventricular diastole by the stream of blood regurgitating into the ventricle, all modern reed instruments have the reed *facing into* the fluid (air) stream This is true even in the case of the mouth organ, or harmonica A blade of grass blown as it is held in the hands, is a somewhat better analogy, although obviously different because the fluid stream passes on both sides of the vibrating element In 1886 Sansom (1344), who compared the murmur of calcific aortic stenosis to the whistle produced with the lips, suggested the Jew's harp as a proper analogy *for the retroverted cusp Suggested* also by Banks (49) in 1857, it is not entirely satisfactory, either, because of the difference in compliance of the reed and in the mode of production of vibration Although the analogy to reed instruments does not fit precisely, it seems adequate for characterizing this group of musical *murmurs*

VIOLIN GROUP Musical extracardiac, i e , pericardial or pleuropericardial murmurs are members of this group (1167) The bowed string instruments function by the general principle of "stick and slip" (1238) (see p 136) The violinist puts rosin on his bow to insure that as he draws *it across the strings it "sticks"* with slight displacement of the strings, then, when a certain limit of displacement is attained "slip" occurs and the string springs back toward and past its position of rest Thus the string is set into, and maintained in vibration Commonly occurring undesirable examples of "stick and slip" include the creaking of hinges shattering of window sashes squealing of tires as a car rounds a corner

Musical pericardial friction murmurs, either acute and transient or more persistent in *nature* occur much less frequently than the noisy variety However, when such occur *one is forced to con*clude that the roughening of the pericardium is arranged in a more orderly manner than usual Possibly the generator area is put into vibration as a whole, in part because it is of limited size rather than there being a great many *individual* small sound generators operating each at a dif*ferent frequency* as may be in effect the case with a generalized pericarditis

"FLITTER" GROUP In this category which cannot be analogized to any of the standard *musical* instruments the mechanism appears to be "flutter" *a phenomenon which Rodbard* (1289 1291 1299) has in recent years analyzed in relation to the cardiovascular system (Fig 92) It is the type of sound produced by blowing through a Sidd valve (Fig 93) the Bronx cheer has a similar

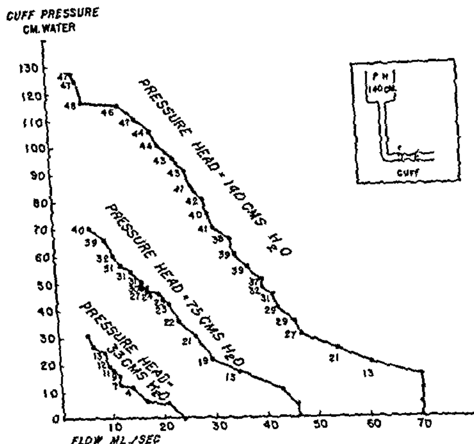


FIG. 92 The flutter phenomenon

Flow through a soft walled tube using pressure heads of 140, 75 and 33 cm. of water respectively. The horizontal axis gives the delivery in ml. per sec. The vertical axis represents the degree of constriction of the tube induced by the air pressure in the glass chamber surrounding the soft walled tube. The numbers adjacent to the three lines represent the repetition rate of closing and opening of the tube as determined by stroboscope.

The upper trace shows the change in delivery as the constriction (cuff pressure) is modified in a system with a driving head of 140 cm. of water. No effect on flow (70 ml. per second) was seen at cuff pressures from zero to 20 cm. of water. As the cuff pressure increased to 25 cm. of water, the flow fell to 60 ml. per sec. and the wall was observed by stroboscope to be closing repetitively 13 times each second. Increases in cuff pressure further reduced flow and increased the rates of closure in a fairly consistent fashion. When cuff pressure was approximately equal to pressure head, flow fell below 5 ml. per sec. and closing and opening of the tube was no longer apparent. Similar data for pressure heads of 75 and 33 cm. of water are also given. The data show that the repetition rate becomes faster with either increasing pressure head or increasing degree of narrowing. (From Rodbard (1213).)

mechanism. The bronchial tree in asthma is a better example on the basis of which to describe the phenomenon. When an elastic walled tube such as a bronchus is narrowed to a critical degree the rapid movement of fluid (air in the case of the bronchi) through the narrowed area causes, by the principle of Bernoulli, such a drop in pressure that the wall at the site of the constriction is pulled in and the lumen further narrowed. But two centrifugal forces tend to enlarge the lumen, the elasticity of the wall and lateral intraluminal pressure. The result of these two opposing forces, one centripetal, one centrifugal, is that the wall is caused to vibrate in a regular periodic and musical manner. In the arterial and venous systems a situation comparable to that in bronchial asthma probably occurs occasionally so that a musical murmur results.

Certain interrelationships of pressure gradient, flow, dimension of stenotic orifices and murmurs

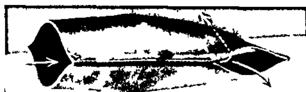


FIG 93A The *Sadd valve* used in spirometers and other respiratory equipment

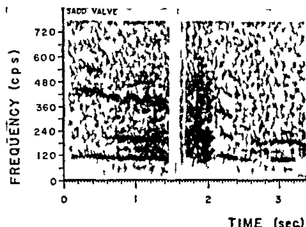


FIG 93B Partially musical partially noisy sound produced by blowing through Sadd valve

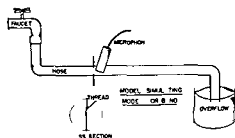


FIG 94A Model simulating the murmur producing properties of aberrant tendons of the ventricle

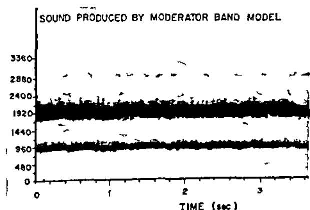


FIG 94B Murmur recorded from the model shown in the last figure

In schematizing circulatory models of stenosis, Burger, van Brummelen and Dannenburg (197) investigated pulsatile flow and concluded that the application to the heart of a formula for constant flow, as in the case of the Gorlin formula, might not be entirely accurate With pulsatile flow "a kind of hysteresis phenomenon" not found with steady flow occurred (Fig 94) there was more flow for pressure "on the way up," less flow for pressure "on the way down" Qualitatively, the authors suggested that this could be explained by the fact that the turbulence of the liquid would increase with increasing flow As time is required for turbulence to set in, there will be a lag as flow increases causing the flow to be less turbulent than would be the case with steady flow at the same pressure differential The lag in the establishment of turbulence is important and may *explain why a murmur does not occur more* often in pulsatile flow even though the critical Reynolds number may be exceeded momentarily

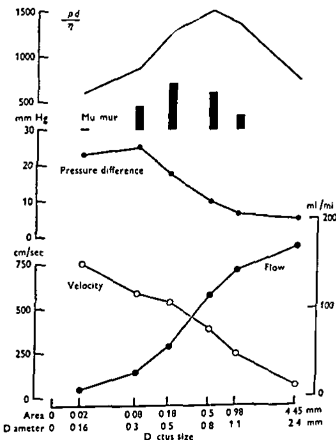


FIG 95 The relationship between the murmur and various hemodynamic parameters in the patent ductus arteriosus of the newborn lamb (From Dawes *et al* (338))

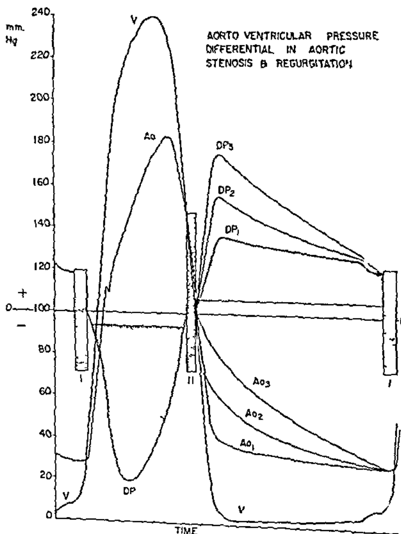


Fig. 36 Pressure gradient and murmurs in aortic valve disease

The curves displayed here are intended to illustrate certain basic relationships in the genesis of the murmur of
aortic stenosis and the murmur of aortic regurgitation The three main curves are those of ventricular pressure (V)
aortic pressure (Ao) of which three alternative curves in diastole are presented and the differential pressure (DP)
of which the three curves corresponding to the three alternative aortic diastolic pressure curves are presented A
basic premise is that the pressure differential across an orifice is intimately related to the intensity frequency char
acteristics of the murmur produced From what is known about the shape of the ventricular and aortic pulse pres
sure curves in aortic stenosis it is evident that the curve of pressure differential in systole will have the shape shown
in the diagram The murmur of aortic stenosis has a curve similar as to intensity and peak frequency (the so-called
diamond systolic murmur of conventional oscillographic phonocardiography and the Christmas tree murmur
of spectral phonocardiography) The configuration of the diastolic portion of the differential pressure curve helps
elucidate several features of aortic diastolic murmurs as they are encountered in practice Since there is likely to
be a certain threshold of differential pressure which must be exceeded before murmur is created the occasional in
fact rare demonstration of a gap between S_2 and the beginning of the diastolic murmur (860) is afforded explana
tion Similarly at the end of diastole the diastolic murmur sometimes stops abruptly with the P wave of the electro
cardiogram or with the atrial heart sound This phenomenon is probably caused by fall of differential pressure below
the murmur threshold as a result of the increment of intraventricular pressure produced by atrial contraction
Within limits the larger the anatomic leak at the aortic valve the lower the differential pressure and the smaller
the diastolic murmur (on first thought a paradoxical phenomenon)

Left heart catheterization is providing actually recorded curves of this type for aortic stenosis and for mitral
stenosis (16, 5 0)

—at the peak of ventricular ejection, for example The faster the rate of cycling of pulsatile flow, the greater was the hysteresis phenomenon

Other findings of the study by Burger and colleagues (197) included the demonstration that, using stenoses with sharp edges, flow was larger with the same pressure difference if the constriction was longer within limits, e g , 0 2, 1 0 and 2 0 cm Flow was maximal in the case of a constriction with sloping profile

In the study of the parameters of flow in relation to murmurs, Dawes, Motte and Widdicombe (338, 339) have made use of the fact that the ductus arteriosus does not close for some days in the newborn lamb The area of the ductus the pressure difference between the aorta and pulmonary artery, the intensity of murmur and Reynolds number were calculated (Fig 95) As might be expected, flow increased steadily with increase in size of the ductus, and pressure difference and velocity of flow fell Although both the intensity of the murmur and the Reynolds number showed a peak in the mid range of ductus size the curves did not coincide The peak for the murmur curve lies on the side of smaller ductus size—and, of course, of greater velocity It is likely that a factor in addition to, or conceivably instead of turbulence is operating in production of the murmur The other factor may be the effect of a jet from the ductus impinging on the opposite

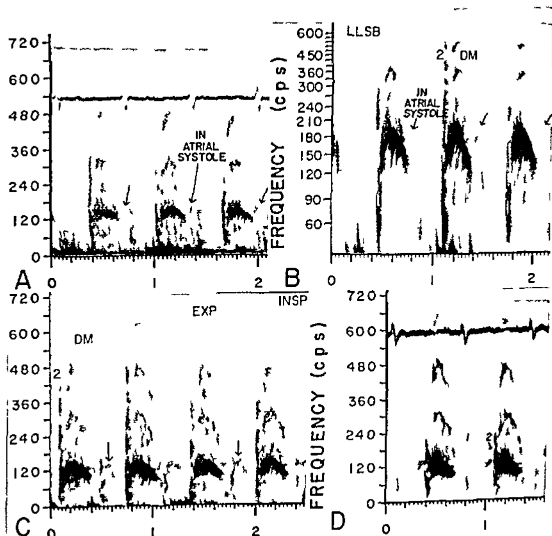


Fig 97 Relation of shape of aortic murmurs to that of aorto ventricular differential pressure curve The se are all recordings made at different times from the lower left sternal border in a patient with a retroverted aortic cusp and aortic *regurgitation on the* basis of syphilis The harmonics of the musical diastolic murmur show the crescendo decrescendo pattern displayed also in the curve of aorto ventricular pressure differential (Fig 96) Note that the musical murmur comes to a halt with atrial systole (e g *D*) or is greatly changed (e g *A*) There is a slight harmonic in early systole in *C* and in late systole in *D* *B* is a logarithmic display

Fig 98 Change in aortic diastolic murmur with change in aorto ventricular pressure differential

Here are displayed recordings (from the same patient as in Fig 9) made at LLSB in the recumbent position before (A) and after (B) the administration of l nor-epinephrine intravenously and in the standing position before (C) and after (D) the administration of hexamethonium intravenously These recordings and displays were made in as identical a manner as possible except for the differences noted In E (see next fig) is presented a synthesis of the findings

Both intensity (as demonstrated by the number of harmonics which are evident) and the frequency level of the harmonics is directly related to aortic pressure The velocity of regurgitation and therefore the frequency of the murmur is raised or lowered with corresponding changes in aortic pressure The interruption of the murmur with atrial systole is most distinct when aortic pressure is highest

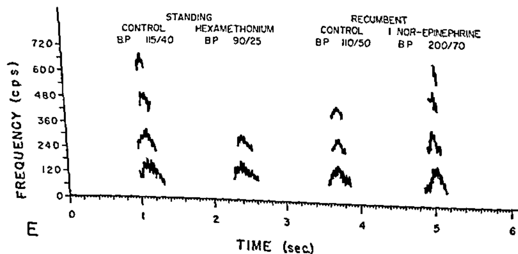


Fig 98E

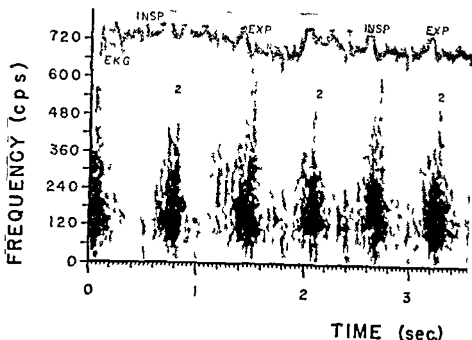


Fig 99 Typical patent ductus arteriosus
LLSB in A K (757481) 6 year old female showing the typical continuous murmur built around S_2

wall This is the collision murmur (*Anprall geräusche*), of which Bondi (131) wrote and which is discussed earlier (p 134)

A very important factor in determining the shape of a murmur, either its time intensity shape in the oscillogram or its time frequency shape in the spectrogram, *is the time course of the pressure* gradient across the orifice where the murmur is generated The relationship between pressure gradient *and shape of murmur is illustrated for* the murmurs of aortic stenosis and regurgitation by the sketch in Figure 96 Figures 97 and 98 illustrate changes in the shape of the diastolic murmur when the gradient changes during the cardiac cycle or is altered by pharmacologic means

In the case of aortic or pulmonary diastolic *murmurs, I have several times observed* (e g, Figure 261) abrupt cessation of the murmur at the time corresponding to atrial systole as indicated by the P wave of the electrocardiogram In some cases a faint transient has occurred at the termination of the murmur This might be termed a reclosure sound A possible explanation for the phenomenon is that ventricular pressure

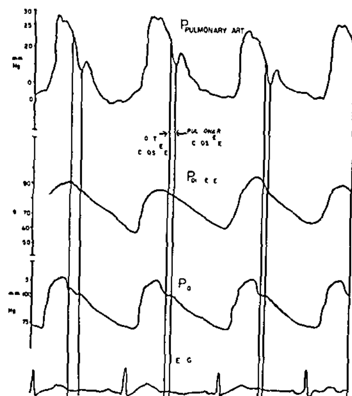


Fig 100 Aorto pulmonary pressure differential in a normal individual

The top and lowest pressure curves are traced from actual recordings of pressure made simultaneously in *the pulmonary artery and aorta in a 20 year old male* with a normal cardiovascular system The middle pressure curve *is derived it is the difference in pressure* between the other two curves In relation to the time of closure of the aortic and *pulmonic valve the peak* occurs late in systole but not as late as one might pre*dict on the basis of the shape of the murmur of patent* ductus arteriosus (Courtesy of Dr Alfred Fishman New York City)

is sufficiently raised by atrial systole that the aortoventricular pressure differential falls below the critical threshold necessary to maintain the murmur. Another possible explanation is that atrial systole by some mechanism influences the position of the aortic valve cusps so that competent closure is possible although it was not previously. The change which might occur with development of atrial fibrillation would shed some light on the question of whether atrial systole is indeed responsible.

Another illustration of the relationship between pressure gradient and shape of murmur is provided by patent ductus arteriosus. In the usual case there is a continuous murmur (Fig. 99) which has its maximal intensity and frequency span late in systole or in the vicinity of the second heart sound (see p. 400). The pressure gradient in such cases likewise demonstrates a peak in late systole (Fig. 100).

There are now available good recordings of the gradient across the mitral valve with mitral stenosis. Again the time course of the gradient bears a close parallel to the shape of the resultant

LEFT ATRIUM

LEFT VENTRICLE

40 25 15 10 0

FIG. 101A. Pressure curves recorded during thoracotomy in a patient with mitral stenosis. Equisensitive recording systems have been used for ventricular and atrial pressure. The diastolic gradient is represented by the distance between the two curves. (Courtesy of Ernest Craige, Chapel Hill, N. C., and of the *New England Journal of Medicine* (313).)



FIG. 101B. A derived graph of the pressure gradient across the mitral valve. The shape of the diastolic murmur in mitral stenosis corresponds closely to that of the curve of pressure gradient. For example, compare the diastolic murmur as represented in Figure 101A and 275.

murmur (Fig 101) In cases of holodiastolic murmur from pulmonary hypertension Davidsen (330A) has shown that there is an appreciable gradient between the pulmonary artery and right ventricle throughout diastole

Velocity of flow, a significant consideration from the point of view of Reynolds formula and also of jet impact in the generation of murmurs, is proportional to the square root of the pressure gradient The relation of pressure gradient to the cross sectional to the valve orifice is indicated in Gorlin's formula (p 286) The pressure gradient varies inversely with the square of the cross section of the valve In terms of the volume of flow, the size of a regurgitant orifice is probably more important than the pressure gradient There is a direct relationship between the volume of flow and the size of the orifice, there is a square root relationship between flow and pressure gradient Gorlin (574) described studies in a patient with the combination of systemic hypertension and mitral regurgitation Reduction of systemic blood pressure to one half its pre treatment level effected only a 20 per cent reduction in the volume of mitral regurgitation In accord with the above observations is the usual failure of murmur intensity, which is probably related particularly to pressure gradient, to indicate the magnitude of regurgitant flow, which is related particularly to orifice size, in mitral and aortic regurgitation

CHAPTER 11

The Transmission of Sound in the Human Body

Alteration in the character of cardiovascular sound in the process of transmission to the surface of the body is inevitable. Francis Bacon (1561–1626) did a famous experiment illustrating this fact. He showed that when he put his head under water in an overturned bucket with air trapped under it, speech sound as heard outside was strangely altered in character (1532). In the chest the situation is even more complex than in Bacon's experiment. There is absorption, reflection, refraction, selective attenuation and conduction at different velocities along various pathways. Clearly the transmission of murmurs determines to a considerable extent their geography, i.e., the distribution of the areas of audibility on the surface of the chest.

The following statements on the transmission of sound in the human body are based largely on empiric observations.

1 Lung is a poor conductor of cardiovascular sound.

2 Bone is an excellent conductor. The observation (589) that very loud heart murmurs audible over the olecranon are not affected by the inflation of a blood pressure cuff on the upper arm is probably evidence of bone conduction. Conduction of the murmur of mitral regurgitation to the sacrum (1478) when the left atrium impinges on the spine may be a similar example.

3 In general, higher frequency components are attenuated more in transmission than are low frequency components.*

4 Components at a level of frequency in the general range of the natural frequency of the thorax are best transmitted. The natural frequency of the normal adult male thorax is in the vicinity of 120 c.p.s., that of the adult female thorax somewhat higher on the average, and that of children's thorax yet higher. (See p. 477.)

5 Groedel and his collaborators (135, 600, 602, 603) found that the normal heart sounds inside the thorax have a sonorous character but outside of the chest wall a metallic high pitched appearance (173). They concluded that the chest wall adds components of higher frequency to the heart sounds, that is, that part of the higher frequencies in the heart sounds are induced oscillations in the ribs. The conclusion was based on observations in cases of defect of the bony thorax and on auscultation in pneumothorax and in the neck.

6 Sounds, particularly murmurs, may be transmitted to the direction of the blood stream at the rate of the pressure pulse wave. The classical example is the murmur of aortic stenosis. According to the views of Kerr and Harp (787) and of Lepeschkin (879) there is in effect a local reproduction of the murmur as the disturbance initiated at the aortic valve is transmitted along the blood vessel (Fig. 102 and 103). Hiestand and Morris (681) noted the transmission of aortic systolic murmurs to the abdominal aorta. Aortic diastolic murmurs, on the other hand, were not transmitted. The Korotkoff sounds appear to be better transmitted downstream than upstream (147). Luisada (978) found that the second Duroziez murmur is transmitted downstream at about the velocity of the pulse wave. The evidence for transmission at the velocity of the pressure pulse is based mainly on timing against the EKG; the onset of the loud murmur of aortic stenosis is at

*See Reference 239 and 969.

loci progressively more distant from the heart. One pitfall to avoid in such an approach being Christmas tree or diamond shaped with a peak in mid diastole, the only part detectable as one progresses away from the heart will be the more intense and progressively later portions of the murmur. However, this source of error cannot account for all the findings reported, particularly those in which the peak of the murmur is used for timing purposes. Leatham (862) published an interesting comparison (Fig 104) of the transmission of the murmurs of aortic stenosis and pulmonary stenosis to the neck. In aortic stenosis the peak is delayed suggesting a prominent mode of transmission in the wall of the aorta, innominate and carotid at a rate in the range of the pulse wave velocity. The peak of the murmur of pulmonary stenosis occurred in the neck about simultaneously with that of the murmur at the precordium, suggesting that the main mode of transmission is more directly through the tissue at a rate closer to that of sound in water. The last mode of transmission is less efficient *viz* the faint audibility of the murmur of pulmonary stenosis in the neck. Many times in very loud murmurs of aortic stenosis one can demonstrate that obliteration of the artery by inflation of a blood pressure cuff does not abolish and often does not even diminish the murmur in loci distal to the cuff on the arm (888). Possibly bone conduction is the main mode of transmission in such cases.

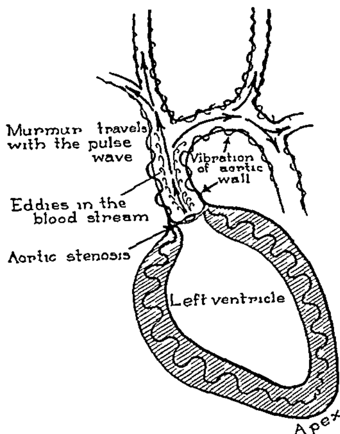


Fig 102 The transmission of the systolic murmur of aortic stenosis from the aortic valve in the direction of the blood flow and backward along the tense contracting ventricular wall to the apex (From Kerr (785))

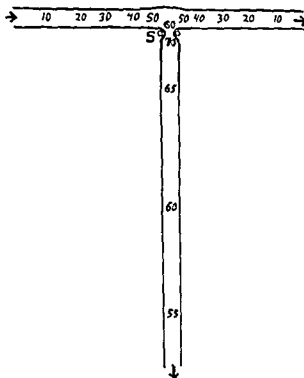


Fig 103 Loudness of murmur in decibels above hearing threshold in the vicinity of a stenosis (S) in a perfused rubber tube. Direction of flow is indicated by arrows (From Lepeschkin (879))

7 Some of the peculiarities in "transmission" of murmurs may in fact be due to peculiar directionality of a jet which actually results in generation of a murmur in the neighbor of the area to which transmission has occurred. See page 134 for many examples. Here I will cite the systolic murmur at the right sternal border in mitral regurgitation and the axillary murmur of aortic regurgitation.

In mitral regurgitation the radiation of the murmur to the apex and axilla is possible contrary to what one might anticipate from the orientation of the regurgitant stream. The reason

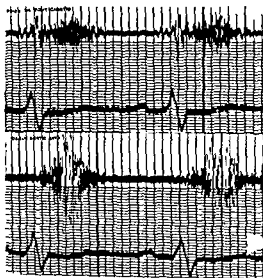


FIG 104A Phonocardiogram of patient with congenital pulmonary stenosis showing synchronism of systolic murmur at the pulmonary area and the right carotid The carotid tracing was taken with increased sensitivity

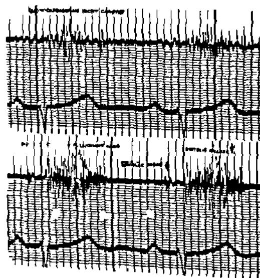


FIG 104B Phonocardiogram of patient with rheumatic aortic stenosis demonstrating delay in the peak of the systolic murmur at the right carotid artery as compared to the aortic area

for the apical radiation has long been a matter of conjecture and to some extent experiment (148) De Sautelle and Carey (347) *thought* that the insertion of the chordae tendineae and papillary muscles might determine the *radiation* that they might act as special conductors I am more inclined to the view that the fact that the left ventricle is in contact with the chest wall whereas poorly conducting lung separates the posterior aspects of the heart from the surface explains the radiation without specific reference to the papillary muscles In 1945 Lian (911) practiced esophageal auscultation with the particular purpose of determining whether the systolic murmur of mitral regurgitation was unusually loud at this site He found it was If sufficiently loud any murmur might be audible in the esophagus but that of mitral regurgitation was usually the loudest Lian thought that this method permitted him to exclude mitral regurgitation as the cause of certain murmurs particularly the late systolic murmur introduced by a systolic click (see p 240) Lian's findings are in agreement with those of intracardiac phonocardiography Lunds and Lu (987) report that a murmur is loudest in that chamber into which the flow of blood is occurring e g the left atrium in mitral regurgitation

In transmission to a locus distant from the area of generation a murmur may be appreciably changed in frequency pattern and therefore in approximate pitch and certainly, quality A striking example is the murmur of aortic regurgitation which may sound quite different—*in particular* lower pitched—when heard at the apex and in the axilla (Fig 105) In part this may be the result of greater attenuation of *higher frequencies* in part a preferential transmission (actual accentuation) of components *in the same* frequency range as the natural frequency of the thorax

There is an interesting difference in the frequency pattern of the murmurs of mitral and tricuspid stenosis In the murmur of *tricuspid* stenosis one usually discovers in the SPCC components extending up to *a frequency* of 400 c p s and occasionally as high as 800 to 900 c p s —a range quite unfamiliar in mitral stenosis Since left atrial pressure *on* an average attains higher levels in mitral stenosis than does right atrial pressure in tricuspid stenosis it is reasonable to assume that even higher frequencies are *generated* in the instance of mitral stenosis However, these *components* of higher frequency in the murmur of mitral stenosis do not reach the surface of the thorax requiring as it does tran

loci progressively more distant from the heart One pitfall to avoid in such an approach being Christmas tree or diamond shaped with a peak in mid diastole, the only part detectable as one progresses away from the heart will be the more intense and progressively later portions of the murmur However, this source of error cannot account for all the findings reported, particularly those in which the peak of the murmur is used for timing purposes Leatham (862) published an interesting comparison (Fig 104) of the transmission of the murmurs of aortic stenosis and pulmonary stenosis to the neck In aortic stenosis the peak is delayed suggesting a prominent mode of transmission in the wall of the aorta, innominate and carotid at a rate in the range of the pulse wave velocity The peak of the murmur of pulmonary stenosis occurred in the neck about simultaneously *with that of the murmur at the* precordium, suggesting that the main mode of *transmission is more directly through the tissue*

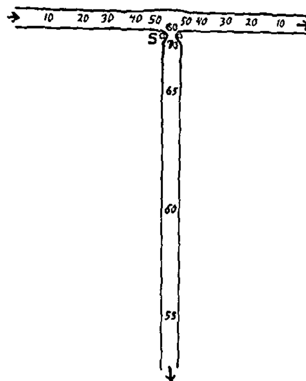


Fig 103 Loudness of murmur in decibels above hearing threshold in the vicinity of a stenosis *(S)* in a *perfused rubber tube Direction of flow is indicated by* arrows (From Lepeschkin (879))

at a rate closer to that of sound in water The *last mode of transmission is less efficient viz*, the faint audibility of the murmur of pulmonary stenosis *in the neck Many times in very* loud murmurs of aortic stenosis one can demonstrate *that obliteration of the artery by* inflation of a blood pressure cuff does not abolish and often *does not even diminish* the murmur in loci distal to the cuff on the arm (888) Possibly bone con*duction is the main* mode of transmission in such cases

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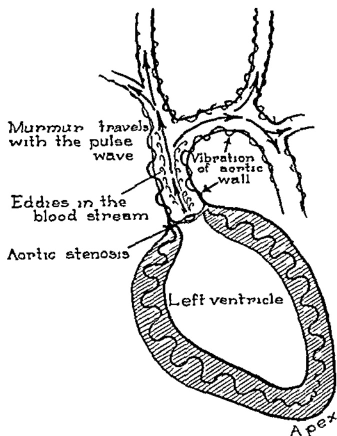


Fig 102 The transmission of the systolic murmur of aortic stenosis from the aortic valve *in the direction of* the blood flow and backward along the tense contracting ventricular *wall to the apex* (From Kerr (785))

difficulties Placing a speaker against the patient's back cannot be practiced as a calibrating procedure since circumferential transmission of the sound in the ribs occurs rather than transmission through the chest which would be of more pertinence Having the patient hold a small speaker in the mouth might be worthy of exploration A calibrating signal in the esophagus would be more valid but has obvious practical drawbacks Marked attenuation of the esophageal signal with emphysema despite relatively little attenuation of the signal applied to the back was found by Lepeschkin The esophageal signal gave results more comparable to those observed with the heart sound

Lepeschkin (879) points out that an analogy between the transmission of murmurs and the carrying of a distant sound by the wind is untenable The velocity of sound in air is about 330 m per sec and a moderately strong wind of 10 m per sec would reduce the distance traveled by the sound per second by about 3 per cent In the case of blood however the systolic velocity is at the most 0 4 m per sec while the velocity of sound in water is around 1400 m per sec this would lead to a reduction of only 0 03 per cent in the distance traveled by the sound

In a T tube experiment (Fig 103) Lepeschkin (879) provides foundation for the view that the murmur is in effect re created in transmission down an elastic tube A murmur is created by a stenosis at point A Arrangements are made for the outflow at C to be equal to the outflow at D therefore velocity of flow is the same in the arms AC and AD of the tube The fact that attenuation is the same in arms BA and AC indicates no assistance to transmission by the flow of fluid The fact that attenuation is so much greater in section BC than in section AD suggests recreation of the murmur at successive sites down arm AD as the flow disturbance originating just beyond the stenosis passes down the tube

This discussion of transmission of cardiovascular sound in the body is obviously on a descriptive and superficial plane Von Gierke and collaborators (1497) have indicated that there are three modes of propagation of sound in the body shear waves compression waves and surface waves There are beginnings on a comprehensive description of the physics of vibrations in living tissues

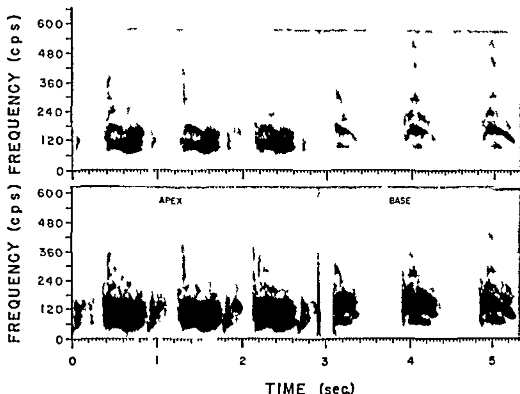


Fig. 105 Preferential frequency transmission in murmurs

Here are presented on the left a recording from the apex and on the right one from the base of a patient with a sea gull diastolic murmur caused by retroverted aortic cusp. Although no attempt was made to record and display the murmurs with identical gain, the relative intensity of the harmonics in the murmur at the apex as compared with those at the base (nearer the site of origin of murmur) makes it evident that those harmonics in the 120 c p s range are preferentially transmitted to the apex. The patient in this case was a 45 year old male. The net natural frequency of the male thorax is in the vicinity of 120 c p s

mission through much more cardiac and other tissue than does the murmur of tricuspid stenosis, in which case the generator area immediately underlies the point of auscultation with much less intervening tissue.

The rate of transmission of cardiovascular sound in body tissues, that is, the transmission of any sound with frequency composition at the lower end of the acoustic spectrum (564), is not established to a completely satisfactory degree. On first thought, sound might be expected to travel in the body at the same rate as sound in water (about 1400 m per sec). However, as mentioned above, evidence is presented by Kerr and Harp (787) that the rate of transmission is more nearly of the order of the velocity of the pulse wave (about 5 m per sec)—a striking discrepancy indeed!

Wood (1586) writes as follows

When liquid is contained in an elastic tube, the yielding of the tube lowers the velocity of sound below the ordinary value for sound in a given fluid. This was demonstrated by early experiments of Wertheim, predicted by Helmholtz and quantitated by Sir Horace Lamb.

Although transmission of sound in the body is not strictly analogous to the situation in an elastic tube, the "yielding" of the tissues has a similar effect. Although not mentioned by Wood, Stewart (see p. 45) also noted the relatively slow transmission of sound in an elastic tube (1351). Lamb devised a formula for predicting the velocity of sound in an elastic tube taking into account such factors as Young's modulus of the wall material and the true velocity in the liquid.

Calibration of the intensity of heart sound and murmurs should take account of differences in the sound transmitting properties of the thorax from individual to individual. Obesity, dimensions of the rib cage, pulmonary emphysema and other factors introduce large variables. Efforts to take the factor of the chest into consideration meets

SECTION III

The Auscultatory and Physical Characteristics of Major Categories of Cardiovascular Sound

CHAPTER 12

The Transients

CHARACTERISTICS OF THE HEART SOUNDS

The *first heart sound* is usually a duller more prolonged sound than the second. It is loudest in the tricuspid and apical area. It can be shown to be made up of at least two components, one related to mitral closure and one to tricuspid closure, usually in that sequence (Fig. 106). The duller quality of the first sound is usually represented in the SPCC by a relatively narrow frequency span with few high frequency components and relatively great intensity of the components at the lower end of the frequency scale (Fig. 107, 108, 109 and 110).

Table 6 presents information on the relative energy content at various levels of frequency of the normal first heart sound. These data are those of Williams and Dodge who published in 1926 (1566). The findings of spectral phonocardiography are in rough agreement, as are also the data of Richards (1269) who used a heterodyne analyzer which produces charts of intensity versus frequency.

The *second sound* is sharper in quality. It is loudest in the aortic and pulmonary areas. Before a person reaches the age of 20 or 30 years the second sound in the pulmonary area usually exceeds that in the aortic area ($P_2 > A_2$), whereas the converse ($A_2 > P_2$) is usually the case *in later life*. The second sound in the aortic area normally is unitary; it is related only to aortic valve closure (Fig. 106). The second sound in the pulmonary *area normally* is a composite of two transients related to aortic and pulmonary valve closure, usually in that sequence if they are not precisely synchronous. Splitting of the second sound is likely *to be heard best in* the pulmonary area and elsewhere along the left sternal border. The second sound at the apex is usually only aortic closure sound.

There is a large body of evidence bearing on the constitution of the second sound beginning at least as early as Ewart in 1896 (445, 446). Wiggers (1548) presented evidence for the predominantly aortic origin of A_2 and M_2 in the form of observations on the changes in the heart sound with various physiologic and pharmacologic maneuvers.

Various onomatopoetic devices for the heart sound, of which lubb tup (or some slight variation) is the most familiar, and various other imitations of the heart sound have been invented. Palfrey (1176) imitated the heart sound by tensing a pocket handkerchief—a long strip for the first sound, a shorter strip for the second (360). Larned (841) devised an ingenious method of putting one hand over one ear and by a combination of taps, strokes and scratches on the back of this hand imitating heart sounds and murmurs for teaching purposes.

The *third heart sound* (Figs. 111 and 112) can be detected in the majority of individuals in the first two decades of life. It is loudest (176) at LLSB and apex, in the decubitus position, especially the primo-decubitus position (first after lying down), during inspiration, following exercise of the legs with the subject in the recumbent position. All the factors enumerated favor venous return of blood. Boyer, Eckstein and Wiggers (146) found the sound in 4 per cent of normal young subjects seated but in 26 per cent recumbent. Sloan and colleagues (1397) reported that the sound was detected stethoscopically in 9.8 per cent of university students but could be demonstrated in 100 per cent by linear phonocardiograms and 39 per cent by logarithmic phonocardiogram. Sloan and Wiehart (1399) found that the physiologic third sound of the dog was diminished or abolished by reduction of venous inflow to the heart.

Fig. 109. Normal heart sound.

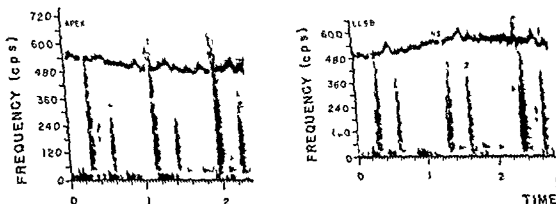


Fig. 110. Heart sounds in J. J. (416[illegible]) normal 16 year old male. At the *apex (left)* only one element of S_1 is demonstrated. This is presumably the mitral closure sound. It corresponds to the first of two *elements of* S_1 which are visible at LLSB (right) and *presumably* represent mitral and tricuspid closure in that *order*. It is possible, however, that the second component of S_1 shown in Fig. 110B is an arterial valve opening *sound*.

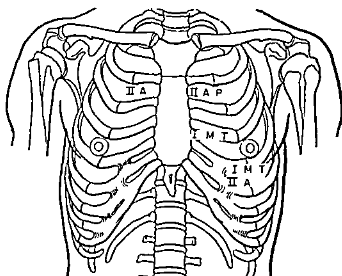


Fig 106 Constitution of normal heart sounds Usually what is heard for second sound in the aortic area is exclusively aortic closure sound whereas in the pulmonary area one hears aortic and pulmonary closure in that order if there is any separation of the elements Usually in normal adults only the aortic closure sound is transmitted to the apex The first sound appears to be a composite of mitral and tricuspid closure usually in that sequence if there is any separation (After Leatham (859))

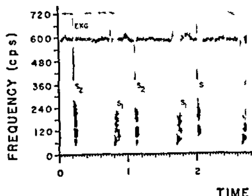


Fig 107 SPCG of heart sounds at apex in a *normal* 32 year old male

The third heart sound is normally dull in quality and has low frequency content in the SPCG

The normal atrial, or *fourth, heart sound* can be detected by stethoscopy in only a minority of individuals and these in the youngest age group It, too is low pitched It is usually best heard at the left sternal border In addition to the exaggerated form of the atrial sound in presystolic gallop atrial sounds are commonly audible in complete heart block and sometimes in atrial flutter Atrial heart sounds are heard particularly often when complete heart block and atrial flutter are present in combination Bennett and Kerr (87) described alternation in the intensity of the atrial sounds in a case of the latter type In cases of complete heart block the occurrence of atrial systole independent of ventricular systole provides an opportunity of demonstrating two components of the atrial sound the first seems to be related to tensing of the atrial wall itself and the second is probably produced in the ventricular myocardium in response to atrial systole (One or both of these elements may have a contribution from the AV valvular mechanism) Weitzman (1523) found that the atrial sound begins 0 12 to 0 17 sec after the onset of the P wave and 0 05 to 0 09 sec after the onset of atrial systole as signalled by the intracardiac pressure recording

Table 7 presents Mannheimer's data (1031) on the frequency content of the four heart sound

Merely for sake of completeness it should be mentioned that a *fifth heart sound* has been described (219), located in diastole after the third heart sound but before the P wave and the

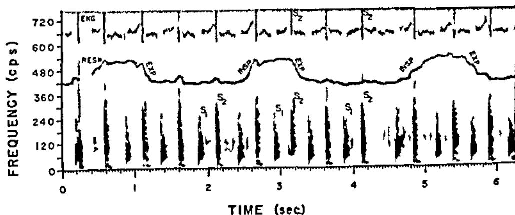


Fig 108 Pulmonary area in a *normal* 5 year old male Note the splitting of S_2 late in inspiration and at the beginning of expiration On the EKG the time of the second heart sound is marked by a verticle line

TABLE 7

Frequency range of normal heart sounds †

Frequency Range	First Sound		Second Sound		Third Sound		Atrial Sound	
	N	Per cent	N	Per cent	N	Per cent	N	Per cent
f								
Below 100	135/135	100	135/135	100	94/135	69 6	74/135	51 8
50-1 5	135/135	100	135/135	100	24/135	17 8	13/135	9 6
100-250	126/135	93 3	134/135	99 3	?/135	1 5	2/135	1 5
1 5-400	10 /135	5 6	1 0/135	93 3	1/135		—	—
250-500	96/135	71 1	124/135	91 9	1/135		—	—
500-1000	40/135	29 6	4 /135	34 8	1/135		—	—

The numbers and percentages listed refer to the cases with components demonstrable in the several frequency band

† From Mannheimer (1088) table 21 p 103

SPLITTING OF THE HEART SOUNDS

Normally the second sound is likely to display it least slight splitting with inspiration (Fig 113) The basis for this is increase in venous return to the right side of the heart due to thoracic aspiration (173) and increased right ventricular stroke volume (1387)[1] The length of ventricular systole parallels stroke volume At the same time that venous return to the right ventricle is increased there is an influence opposite in direction and perhaps less in magnitude on venous return to the left ventricle (1561) Obviously in normal inspiratory splitting of the second sound the sequence is aortic closure sound then pulmonary closure sound With normal continuous respiration in healthy subjects the interval between the two components of S usually does not exceed 0 03 sec in expiration Boyer and Chisholm (147) point out that not only is the pulmonic component likely to be later in inspiration but the aortic component usually occurs earlier as well See Figures 114 and 115 and Table 8 Moss and Johnson (1120) found that the same amount of stretch resulted in a greater increase in the stroke volume of the right ventricle than of the left This result they attributed to the difference in architecture of the wall of the ventricle such that the fibers of the right ventricle are placed more nearly on a linear stretch whereas the stretch is circumferential in the case of the left ventricle On the basis of this experience in the experimental animal one might anticipate that any respiratory fluctuations in filling pressure would have a more pronounced effect on stroke volume and systolic duration in the right

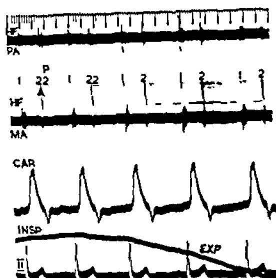


FIG 113 Normal inspiratory splitting of S

Phonocardiograms from the pulmonary and mitral areas (PA and MA) with an indirect carotid tracing (CAR) and electrocardiogram all taken simultaneously *Splitting of the second sound is confined to the pulmonary area and to inspiration (in p)* The earlier component (A_2) is shown to be caused by aortic closure by its synchrony with the *dicrotic notch of the carotid* tracing and is transmitted to the mitral area Later pulmonary closure (P_2) is confined to the pulmonary area Time intervals in the recordings shown here are 0 2 and 0 004 sec HF *refers* to the fact that the characteristics of the phonocardiograph were such as to favor higher frequency components in the sounds (Courtesy of Dr Aubrey Leatham)

[1] Effect of respiration on QRS amplitude and the relation of these effects by change in right and left ventricular stroke volume have been pointed out (834)

TABLE 6

*Frequency energy composition of heart sounds**

Frequency	Total Energy
c p s	*Per cent*
50	56
60	27
70	10
80	4
90	2
100–110	1

* From Williams and Dodge

atrial sound. Calo (221) devotes five pages to a discussion of *the normal fifth sound and two* more to the pathologic fifth sound! We have never observed such a sound in recordings from *several thousand patients. The possibility of a* biventricular gallop, that is, a gallop arising in each ventricle and slightly asynchronous, has not been confirmed (see p. 179). *It is not at all clear* what the vibrations indicated by authors as a fifth sound may be.

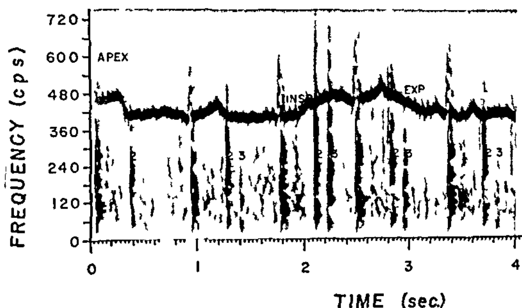


Fig. 111. In this recording from a *normal* 12 year old child (S. W. A96175) inspiration is accompanied by speeding of the rate (sinus arrhythmia) and accentuation of the third heart sound. There is a faint somewhat musical systolic murmur of the type described by Still (p. 244).

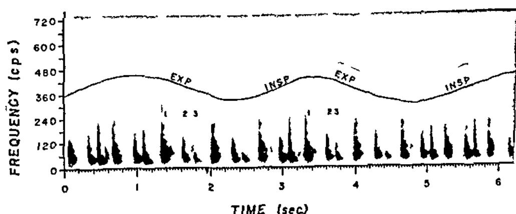


Fig. 112. Speeding of heart rate and accentuation of S_3 with inspiration

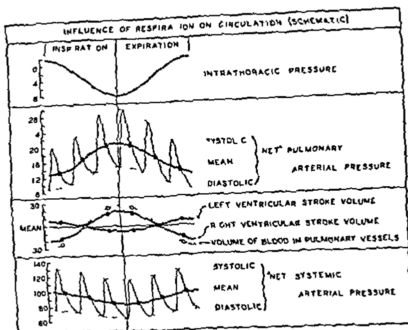


FIG 116 (From Lauson Bloomfield and Cournand (853))

of splitting of the second sound which are appropriately termed by *Leatham (861) mechanical* and *electrical*

In the *mechanical* variety of splitting (Fig 117 118) the basis is a *discrepancy in the stroke* volumes of the two ventricles The classical example—aside from the phenomenon of normal inspiratory splitting—is *provided by* atrial septal defect (Fig 119) Here because of the *left* to-right shunt the right ventricle may pump considerably more blood than the left and take longer in doing so with resultant splitting of S_2

Splitting of the second heart sound of moderate degree occurs commonly in severe mitral regurgitation (Fig 120 and 121) Presumably because of the double route of ejection from the left ventricle aortic closure occurs prematurely That the splitting is no greater than it is may be the result of a cancelling effect of prolongation of left ventricular systole from increased stroke volume The splitting of S_2 in mitral regurgitation is likely to be evident in the pulmonary area where the systolic murmur is less audible Slight *splitting of* S_2 *is* likely to *occur in* ventricular septal defect for reasons *identical* to those in mitral regurgitation the hemodynamics of the two situations is analogous

Contrary to the general impression *hyper*tension of neither the pulmonary nor the systemic circuit causes conspicuous splitting of the second sound Studies of pulmonary hypertension such as the so-called primary pulmonary hypertension have often described close splitting of the second sound *(444 1589)* However the usual temporal relationship of aortic and pulmonary closure was preserved pulmonary valve closure did not occur prematurely as a result of elevation of pulmonary artery pressure It may be that right ventricular systole is longer in such cases The influence of *inspiration* may be less impressive Clinical experience relates splitting of S_2 much more to pulmonary hypertension than to systemic *hyper*tension Any slight tendency to splitting would be less *evident* in the latter case *for reasons* expounded *above* The clinical impression that a widely split P_2 *occurs with* pulmonary hypertension has *probably* been engendered in large part by the easy audibility of the mitral opening snap in the pulmonary area of patients with pulmonary hypertension on the basis of mitral stenosis

Contrariwise hypotension in the pulmonary artery in congenital pulmonary stenosis can result in delay of pulmonary valve closure and splitting of the second sound Since it is the pressure differential on the two sides of the valve

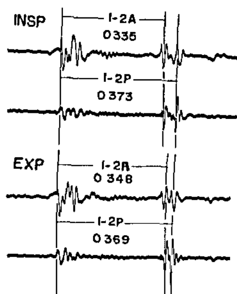


FIG. 114. Typical recording from which the measurements indicated in Figure 115 and in Table 8 were made. With inspiration 1-2 A is shorter and 1-2 P is longer than in expiration. The two recordings of each pair were made with galvanometers of different frequency response characteristics.

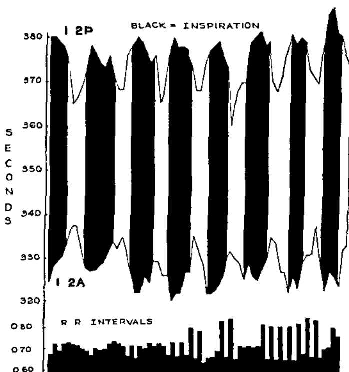


FIG. 115. Variation in splitting of S during quiet breathing in M. M., normal 21 year old male studied in recumbent position. Because of a shifting pacemaker RR intervals were variable but demonstrated no consistent relationship to phase of respiration. The splitting of inspiration is contributed to both by delay of pulmonary closure sound and by earlier occurrence of the aortic closure sound.

TABLE 8

*Changes in the second heart sound during inspiration in fifteen normal subjects**†

	Mean Max 1-2AΔ†	Mean Max 1-2PΔ†	$\frac{1\text{-}2A}{1\text{-}2P} \times 100$	Mean of Split†	Percentage of Splitting Due to 1-2AΔ
Mean	11.5	11.9	97%	20.8	49.3
Range	(7-17)	(5-24)	(70-320)	(11-42)	(33-80)

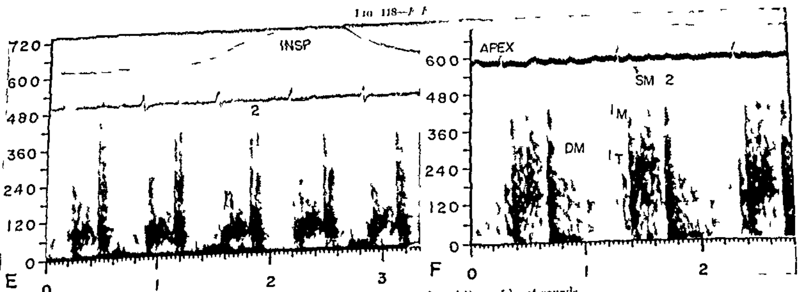
* From Boyer and Chisholm.
† Change (Δ) in milliseconds.

ventricle than in the left. Lawson and co-workers (853) provided the lucid schematic representation of certain hemodynamic interrelationships and respiration, shown in Figure 116.

In teaching students to hear splitting it is helpful to demonstrate what they should be hearing by striking two fingers on the table in a very slightly asynchronous manner. This alternating periodically with synchronous tapping of the fingers serves to convey very well the impression of respiratory splitting.

With labored respiration, with pulmonary fibrosis in which the compliance of the lung is reduced, with airway obstruction, and with any other condition such that an unusually great fall of intrapleural pressure occurs with inspiration, there is likely to be exaggeration of the normal inspiratory splitting of the second sound (1084). In a patient over 40 years of age without evidence of heart disease striking inspiratory splitting of S suggests some form of respiratory disease which is accompanied by exaggeration of the negativity of intrapleural pressure during inspiration. The finding is of most significance if breathing is ostensibly unlabored. Inspiratory airway obstruction as by bronchogenic carcinoma and parenchymal or pleural fibrosis are possibilities in such cases. Inspiratory splitting is a special case of the first of the two main varieties

[2] Another basis for splitting, asynchronous closure of individual cusps of a valve, as proposed by Skoda in 1854 (1395), is unlikely. No corroboration for this idea could be obtained by examining frame by frame available motion pictures of functioning heart valves made at a speed of 24 frames per second. Slight splitting seemingly demonstrated (1077) in the sounds produced by the Hufnagel plastic prosthesis (see ref 1077) may be the result of a filter artifact. There is no evidence that closure followed by "bouncing" and reclosure occurs normally.



Figs. 117 and 118. Mechanical factors in the splitting of heart sounds.

A, pulmonary area in 13 year old child. Slight early systolic murmur. B, apex in 43 year old patient with interatrial septal defect. Each compo-
nent of split S_2 has frequency pattern of a valve closure sound. C, pulmonary area in 50 year old patient with systemic arterial hypertension and
left sided thoracoplasty for pulmonary tuberculosis. Split 1 2 probably caused by exaggerated negativity of intrapleural pressure. Scratchy mid
or late systolic murmur believed to be extracardiac. D, pulmonary area in pulmonary stenosis. Probably valvular type. Note Christmas tree ejec-
tion stenosis murmur and delayed pulmonary closure sound or pulmonary reversal nap (IIp). The systolic murmur appears to continue slightly
after the aortic closure (IIao). E, pulmonary area in 20 year old patient with mitral regurgitation. There is splitting of the second sound which
is exaggerated by inspiration. Note the decrescendo systolic murmur. F, apex in a patient with rheumatic mitral regurgitation. The systolic murmur
begins immediately with the second component of the split first sound which therefore is probably mitral closure sound. If so, it is considerably
delayed and has an anomalous relationship to the tricuspid closure sound. The second sound is unitary—presumably only aortic closure sound—
and is followed immediately by a decrescendo diastolic murmur which probably had its origin at the aortic orifice. Because of the aortic regurgi-
tation it is possible the second component of S_1 is an aortic ejection sound which is usually well heard at the apex.

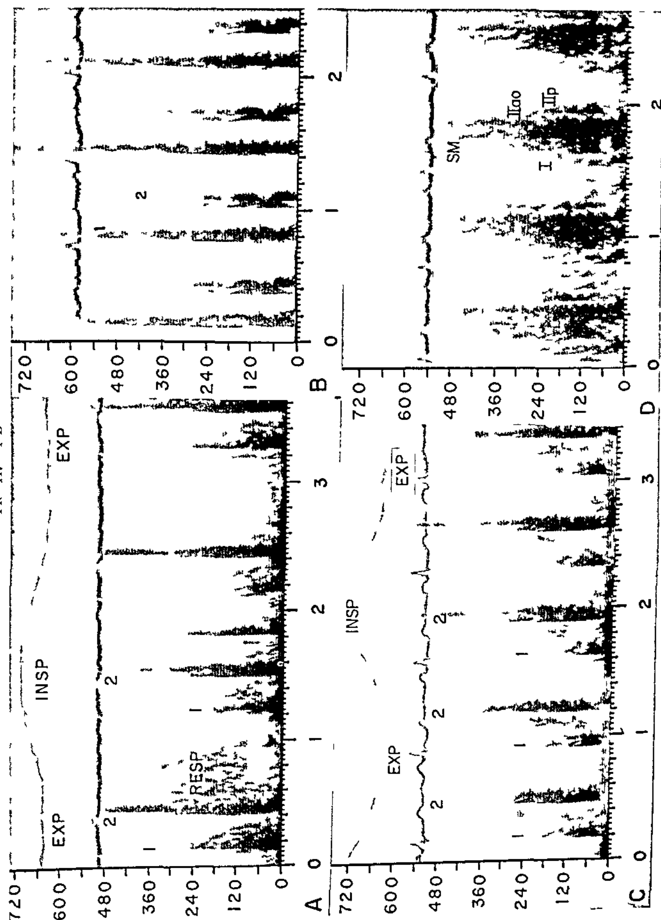


Fig. 117—A-D

FIG. 118—E F

FIGS. 117 and 118. Mechanical factors in the splitting of heart sounds.

A, pulmonary area in 13 year old child: slight early systolic murmur. B, apex in 43 year old patient with interatrial septal defect. Each component of split S_1 has frequency pattern of a valve closure sound. C, pulmonary area in 30 year old patient with systemic arterial hypertension and left sided thoracoplasty for pulmonary tuberculosis. Split I probably caused by exaggerated negativity of intrapleural pressure. Scratchy mid or late systolic murmur believed to be extracardiac. D, pulmonary area in pulmonary stenosis, probably valvular type; note Christmas tree ejection stenosis murmur and delayed pulmonary closure sound or pulmonary reversal snap (IIp). The systolic murmur appears to continue slightly after the aortic closure (IIao). E, pulmonary area in 20 year old patient with mitral regurgitation. There is splitting of the second sound which is exaggerated by inspiration; note the decrescendo systolic murmur. F, apex in a patient with rheumatic mitral regurgitation. The systolic murmur begins immediately with the second component of the split first sound which therefore is probably mitral closure sound. If so, it is considerably delayed and has an anomalous relationship to the tricuspid closure sound. The second sound is unitary—presumably only aortic closure sound—and is followed immediately by a decrescendo diastolic murmur which probably had its origin at the aortic orifice. Because of the aortic regurgitation it is possible the second component of S_1 is an aortic ejection sound which is usually well heard at the apex.

that controls its closure, the effect of pulmonary hypotension is relatively greater by reason of the ventricular hypertension When ventricular systolic pressure is in the range of 100 to 200 mm Hg, it might be anticipated that a longer time will be taken for intraventricular pressure to fall below that in the pulmonary artery

In a sizeable group of cases of pure pulmonary stenosis, Leatham and Weitzman (866) found a direct correlation between the level of right ventricular pressure and the degree of separation of the aortic and pulmonary components of S Although the difference between ventricular and pulmonary pressure is a simple and rather obvious basis for the splitting, the possibility of a slower and more prolonged right ventricular systole as a contributing factor is also present

Gray (587) in writing about paradoxical splitting of the second heart sound—that is, aortic component following pulmonary to produce splitting which was observed mainly in expiration—stated that 10 of 23 cases of aortic stenosis and 10 of 29 cases of patent ductus arteriosus showed the phenomenon In the latter group increased stroke volume of the left ventricle

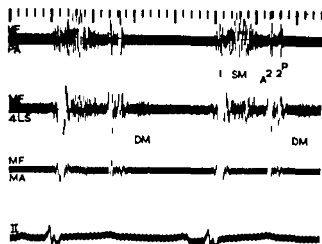


Fig 119 Split S_2 with ASD

The pulmonary closure sound is louder in the pulmonary area the aortic closure sound is louder at the apex There is a systolic ejection murmur resulting from high flow in the right ventricular outflow path and stopping in a characteristic fashion before S_2 Incomplete right bundle branch block is suggested by the form of the QRS in the electrocardiogram (Courtesy Dr Aubrey Leatham)

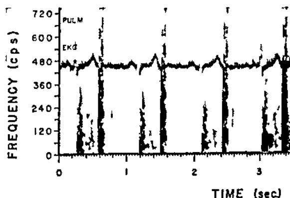


Fig 120 Split S_2 with mitral regurgitation

I M (764492) 19 year old male had at the apex typical auscultatory finding of MR S consistently split

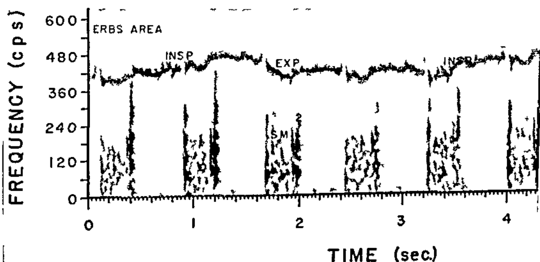


Fig 121 Split S_2 of mitral regurgitation

Erb s area in J Y (763674) 16 year old female with rheumatic mitral regurgitation and moderate pulmonary hypertension An early systolic click is seen in some cycles S_2 is fairly consistently split the pulmonary component is clearly second The splitting is slightly increased with inspiration (See Fig 58 for recording at apex)

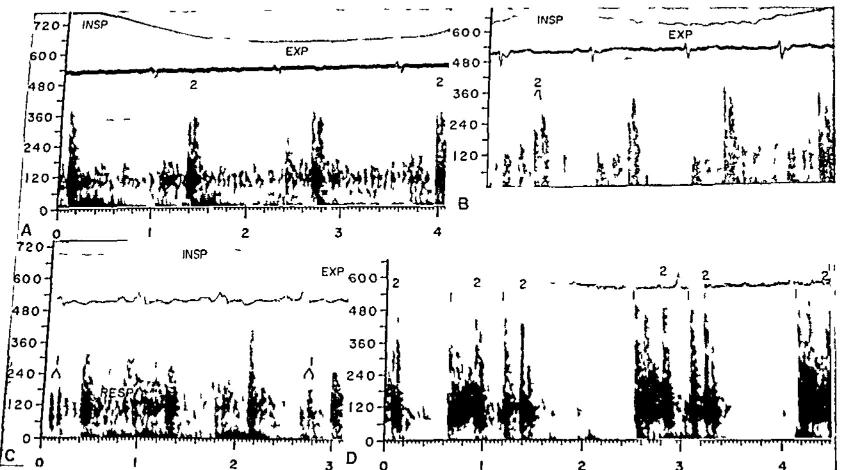


Fig. [illegible] Electrical type of heart sound splitting.

A pulmonary area, left bundle branch block; note paradoxical effect of expiration. *B* pulmonary area, right bundle branch block; note exaggeration of splitting with inspiration. *C* split first sound in left bundle branch block. Although not clearly split, the second sound is wider in expiration. *D* mitral regurgitation; second sound split in normal cycles; both first and second sound split with ventricular premature contractions; note the decrescendo systolic murmur which is holosystolic.

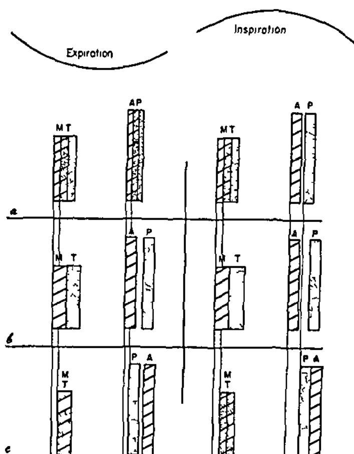


FIG 123 Effect of respiration on the splitting of heart sounds in bundle branch block

(*a*) Normal (*b*) right bundle branch block and (*c*) left bundle branch block Note that with neither type of block is splitting of the first sound to be expected and that splitting of the second sound in left bundle branch block is *paradoxical and of lesser degree than in right* bundle branch block

over the right is the responsible factor The splitting in aortic stenosis has a basis comparable to that in pulmonary stenosis

In the *electrical* variety of splitting of S (Fig 122) the basis is a slower spread of the triggering impulse in one ventricle as compared with the other The classical example is bundle branch block Particularly in right bundle branch block is *there likely to be impressive splitting of S* since the normal tendency for pulmonary closure to lag behind aortic closure is exaggerated by the delay in triggering of the right ventricle The delay in pulmonary valve *closure is further* exaggerated in *inspiration* through an additive effect of normal inspiratory splitting

In left bundle branch block the degree of splitting is likely to be less impressive because the delay in aortic valve closure tends merely to cancel out the normal slight lag in pulmonary valve closure Furthermore, with inspiration the splitting is likely to be least, in left bundle branch block the splitting of S is *maximal in expiration* The reason will be evident after a bit of thought The different effects of inspiration on splitting in the two varieties of bundle branch block are graphically represented in Figure 123 and provide a bedside method for distinguishing the two forms

In both right and left bundle branch block as well as in mitral regurgitation the splitting of the second sounds tends to become fixed when heart failure develops (Fig 124), that is, respiration does not have its usual effects (1218a)

True splitting of S_1 is fairly uncommon in bundle branch block, contrary to the conclusion of King and McEachen (795) who reported it in 28 of 56 cases in a stethoscopic study The frequent association of presystolic gallop and bundle branch block may be responsible for a mistaken impression of the incidence of split S_1 in *bundle* branch block (897) Contro and Luisada (286) found "three groups of vibrations" in the first sound in two cases of left bundle branch block The last group of vibrations in the case they picture is so late as to suggest a great vessel snap ("ejection sound") rather than a true part of S_1 *True splitting of S_1 is more likely to occur in* congenital bundle branch block as an isolated abnormality (Fig 125) This finding accords well with the finding of *Braunwald and his* colleagues (168) that only in this type of case is there significant asynchrony in onset of right and left ventricular systole

Conditions which may simulate splitting of S_1 (Fig 126) are (1) presystolic gallop (919) and (2) the early *systolic click of disease of the great* vessels (see below) Splitting of S may be simulated *by a late systolic click of pericardial origin* (preceding S) and by a mitral opening snap an early diastolic snap of constrictive pericarditis or a protodiastolic gallop (all following S) *Protodiastolic gallop is likely to endow the rhythm* with a cadence rarely encountered with true splitting However the sound of constrictive pericarditis since it is located closer to the second sound is more likely to confuse

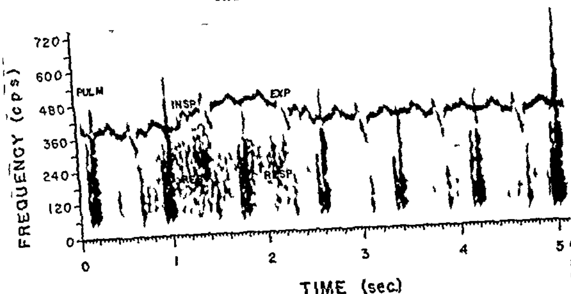


Fig 124 Right bundle branch block without respiratory variation
J D T (6 353) ?? years old has idiopathic myocarditis Possibly because of incompetence of the heart no exaggeration of the splitting occurs with inspiration

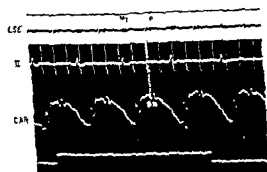


Fig 125 Congenital right bundle branch block
Exaggerated asynchrony of the two components of the first and second sounds resulting in abnormally wide splitting With inspiration (upward deflection of the baseline) there is further delay in pulmonary valve closure and further widening of the second sound Courtesy of Harvey and Ferloff (65?) and of *Circulation*

Because of much overlap of the degree of splitting with that occurring normally splitting is of less diagnostic value in the pathologic states with which it may occur atrial septal defect bundle branch block mitral regurgitation and respiratory disease resulting in exaggerated cyclical variations in intrapleural pressure Splitting has more pathologic significance (1) when it occurs in adults than when it occurs in children (2) when splitting is exaggerated with expiration rather than with inspiration (suggesting the paradoxical splitting of left bundle branch block aortic stenosis or patent ductus arteriosus (3) when there is no clinical evidence of respiratory disease or labored respirations and (4) when splitting persists unaltered throughout all phases of respiration (as usually is the case in ASD) so-called fixed splitting"

FACTORS ALTERING THE HEART SOUNDS

Obviously the heart sounds are louder when the chest wall is thin and fainter when it is thick or when there is pulmonary emphysema or obesity Anatomical changes—in the size of the chambers of the heart and great vessels or in the rotation of the heart—by altering the relation of the various portions of the heart to the chest will cause change in the heart sounds For example idiopathic dilatation of the pulmonary artery is likely to be accompanied by a loud P_2 even though there is no pulmonary hypertension For a similar reason P_2 is almost always accentuated in cases of atrial septal defect even when pulmonary hypertension is absent Enlargement of the right ventricle with atrial septal defect is likely to result in the right ventricle s representing the cardiac apex As a result the pulmonary closure sound is likely to be heard at the apex

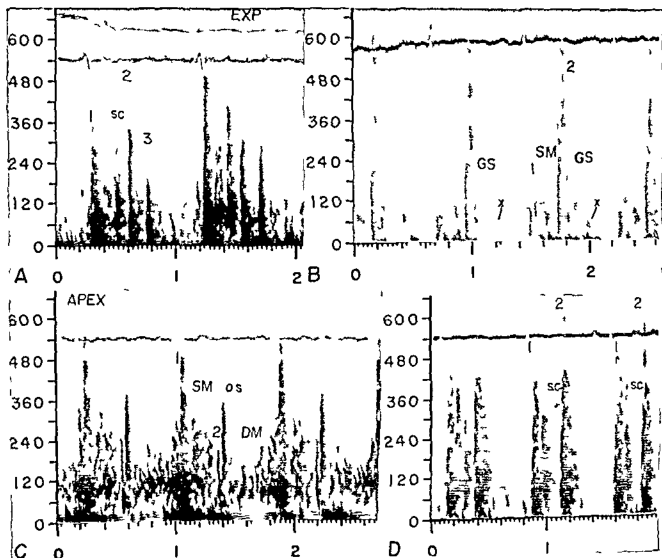


Fig 176 Phenomena simulating split sounds

A late systolic click of about the same intensity as that seen at the apex was also present at left sternal border and pulmonary area There is a history of acute rheumatic fever during which pericarditis was observed four months before this recording third heart sound is also present B this patient with mitral stenosis had considerable pulmonary hypertension with resultant accentuation of the pulmonary closure sound The temporal relationship of the two components of P_2 is not altered There is an exceedingly subtle diastolic murmur (probably Graham Steell) beginning immediately with P_2 There is a faint sound in mid diastolic (which is difficult to identify) at the termination of the Graham Steell murmur It is too early for an atrial heart sound and perhaps too late for a third heart sound It may represent a valve closure sound— slight leak occurs through a small separation of the cusps as long as the pressure differential is sufficient to maintain the separation As soon as the pressure gradient drops below a certain threshold coaptation of the previously separated surfaces occurs with production of an audible sound C recording at the apex in a patient with severe mitral stenosis and a moderate degree of mitral regurgitation note the snapping mitral closure sound the systolic murmur and the diastolic murmur with presystolic crescendo There is seemingly a split second sound Actually it is clear that this is in fact a second sound followed closely by an opening snap This patient also has aortic stenosis The aortic closure sound is so attenuated that it is doubtful that it is transmitted to the apex where the pulmonary closure sound alone is represented That the sound marked OS is indeed mitral opening snap is corroborated by its snapping appearance pure frequency content and elevated frequency bottom D an example of early systolic sound in the pulmonary area caused by dilated pulmonary artery due in this case to atrial septal defect In addition both the first and the second sounds are slightly split

as is also the diastolic rumble of relative tricuspid stenosis

Aside from these extravalvular and extracardiac factors affecting the heart sounds there are valvular factors which can be classified as follows

1 The speed of valvular closure
2 The force of valvular closure

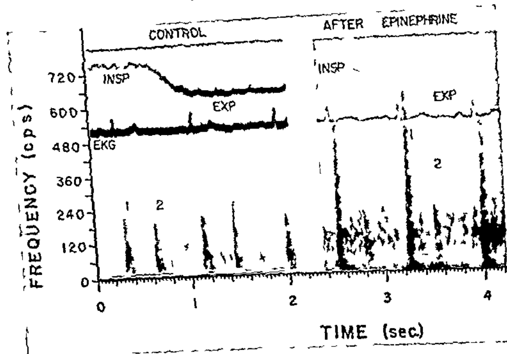


Fig 17 Effect of epinephrine on normal heart sounds

Demonstrated in A are the heart sounds recorded at the lower left sternal border in a normal subject. In B is a recording from the same area made under *identical conditions* of amplification five minutes after the administration of 1.0 cc of epinephrine 1:1000 subcutaneously. *The second heart sound is little changed.* (Systemic blood pressure was 100/70 mm Hg during the control recording and 114/64 mm Hg after epinephrine.) The striking change in the first sound consists of increase in peak frequency (frequency span), appearance of more conspicuous harmonics and increase in over all loudness as indicated by *blackness*. *The first two features account for the impression of snappiness which the first sound after epinephrine conveys to the ear.*

The accentuation of the first sound is probably closely related to the *subjective palpitation* which accompanies epinephrine administration. Increase in the *velocity* of valve closure is probably principally responsible for the accentuation. As is demonstrated in *B*, higher frequencies are produced and it is largely the contributions of these to total intensity which are responsible for the over all intensification.

(Excessive *background* noise appeared in the recording after administration of epinephrine. The amplification during the stages of recording *and* analyzing was identical in the two cases. Several explanations for the increased background noise in the second recording are possible: (1) increase in ambient (*room*) noise, (2) muscle noise from the tension engendered by the drug, (3) vascular noise from circulatory changes.)

3 The excursion in valvular closure

4 The physical quality of the valve

The faster a valve closes the louder is likely to be the sound produced. The youthful heart displays snappy heart sounds for this reason. Furthermore administration of *adrenaline* in doses which produce little or no change in intravascular pressures may abbreviate isometric contraction* (801) and accentuate the first heart sound (Fig 127). The loud first heart sound in thyrotoxicosis probably has the same basis; many of the manifestations of thyrotoxicosis appear to be the result of potentiation of *the effect of catecholamines* by thyroid hormone. *Conversely*, in myocardial disease the first sound may be described as "dull," "of poor quality," "faint." *In this situation* even though intravascular pressures are normal the lower rise in intraventricular pressure makes for a dull sound. Not only is the sound produced by rapid closure of a valve louder but it also has higher frequency content according to a general principle defined as follows by Lamb (833):

"the higher harmonics are excited in greater

*Strictly speaking it is not abbreviation of isometric contraction which is operating in producing the accentuation of the first sound since the AV valves are already closed during this period. However there is usually a direct relationship between the velocity with which they are closed and the duration of isometric contraction.

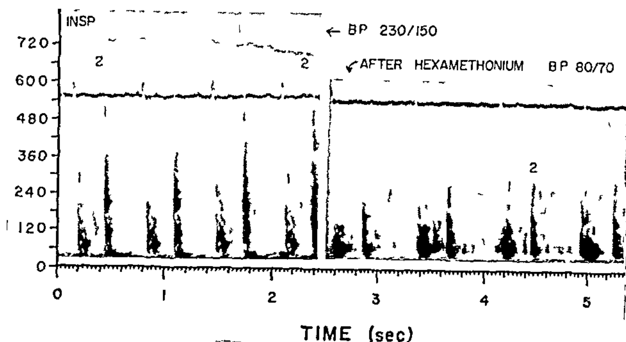


FIG 128 Heart sounds before and after administration of hexamethonium to a hypertensive patient

In this pair of tracings from the left lower sternal border variations in blood pressure were accompanied by little change in the first heart sound which in each instance is split possibly into mitral and tricuspid components At the hypertensive level the second sound is louder (blacker) and has a greater frequency span The variable involved here is *force* of closure Velocity of closure an important factor in the accentuation of the first sound (S_1) in Figure 127 is intimately related to force of closure It is likely that aortic valve closure is more rapid at elevated levels of diastolic pressure

Electrical interference at 240 cycles is demonstrated

relative intensity the more abrupt the character of the originating disturbance"

Accentuation of the second heart sound with arterial hypertension of the systemic or pulmonary circuit is familiar and represents a change related to increase *in the force or pressure of valvular closure* (See Figure 128 for demonstration of the reversal of this effect) Because of the double origin of P' the second sound in the pulmonary area may be louder than that in the aortic area in cases of systemic arterial hypertension, especially in young persons The reason for this is that the aortic component of the second sound has been so accentuated that the combination of closure sounds is louder than the aortic closure sound alone as heard in the aortic area

The first heart sound may be accentuated in association with hypertension of either circuit In this situation increased force cannot be the factor in the accentuation since the valve closes when pressure in the ventricle exceeds that in the atrium and atrial pressure may not be elevated The factor responsible for the accentuation must be increased rapidity of closure If the phase of ventricular contraction preceding opening of the arterial valves is not *prolonged then to attain* the level of diastolic pressure in one or the other of the great vessels the pressure in the ventricle must rise faster than it does normally

In so called "auscultatory alternans" which sometimes accompanies pulsus alternans (p 421) the alternately stronger and weaker heart sounds are dependent on differences in the force of valve closure

The excursion of the valve during closure is another important factor in the intensity of the first sound at least and possibly of the second It has already been pointed out on page 112 that the intensity of the first heart sound in various grades of atrioventricular dissociation (Fig 129) finds interpretation in the influence of atrial systole on the degree of separation of the AV valve cusps and the extent to which the belly of the cusps is displaced toward the atrium The first heart sound is likely to be accentuated in ventricular premature contractions due to the

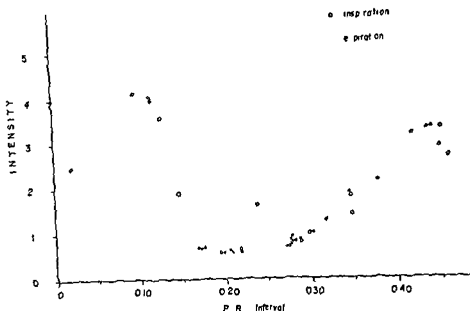


FIG 1.01 The relation of intensity of the first heart sound (in arbitrary units) to the duration of the PR interval (in seconds) in a dog in which complete heart block was produced surgically by the method of Starzl and Gaertner (1434) The intensity of the first sound is presumed to be related directly to the degree of separation of the cusps at the time of contraction of the ventricle With short PR intervals one can infer wide separation of the cusps (or deep displacement of the cusp bellies) caused by atrial systole At longer PR intervals (in the normal range) the cusps would appear to be close together probably caused by the breaking of the jet phenomenon described by Henderson and Johnson (6 0) With still longer PR intervals there has been time for the cusps to return to a more wide open more neutral position Similar curves in children have been reported (1382) (Expiration includes readings in both the expiratory phase proper and the phase of expiratory apnea) (Observation of Dr S H Boyer IV)

occurrence of valvular closure during early diastolic filling when the valve is relatively wide open[4] The tricuspid closure sound in atrial septal defect is likely to be accentuated (see p 349) probably because of a wide open position at the onset of ventricular systole as a result of long continuing ventricular filling In part the accentuated first sound in mitral stenosis may be due to displacement of the bellies of the mitral cusps far into the ventricle because of elevated pressure in the left atrium at the time that the ventricle contracts The short excursion of the aortic leaflets in calcific aortic stenosis is a factor which tends to produce attenuation of the second sound

In this discussion of the relation of valve position to intensity of the heart sound it will be noted that the current thought is that events at the cusp belly rather than at the coapting margin are important in the production of the sounds Therefore when it is stated that the valve is wide open or almost closed it is really the position of the cusp belly that is being referred to Movement of the margin of the cusps of the AV valves may in fact be minimal (1326)

fibrosis and calcification tend to accentuate it With extrasystoles a faint second sound may occur largely because the cusps of the arterial valves are spread minimally The fact that diastolic pressure is lower is also a factor, of course With a large stroke volume it is possible that as Rouanet predicted from his observation in a model (p 40) accentuation of S_2 may occur from wide separation of the cusps of the arterial valves Other clinical examples of this mechanism may be the accentuated pulmonary closure sound in atrial septal defect and the accentuated aortic closure sound in patent ductus arteriosus

A faint first sound is a usual accompaniment of PR interval longer than 0 20 sec as in the first degree heart block of active rheumatic carditis (885 p 123) The intensity of S_1 has been acclaimed as a useful clinical sign for following the progress of patients with rheumatic fever The finding is thought to have its basis in a semiclosed position of the AV valves because of a

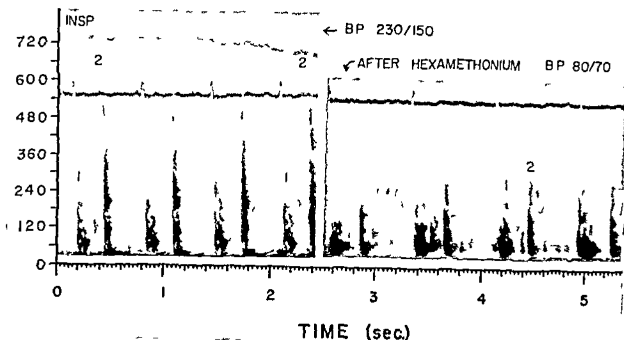


Fig 128 Heart sounds before and after administration of hexamethonium to a hypertensive patient

In this pair of tracings from the left lower sternal border variations in blood pressure were accompanied by little change in the first heart sound which in each instance is split possibly into mitral and tricuspid components. At the hypertensive level the second sound is louder (blacker) and has a greater frequency span. The variable involved here is *force of closure*. *Velocity of closure*, an important factor in the accentuation of the first sound (S_1) in Figure 127, is intimately related to force of closure. It is likely that aortic valve closure is more rapid at elevated levels of diastolic pressure.

Electrical interference at 240 cycles is demonstrated.

relative intensity the more abrupt the character of the originating disturbance."

Accentuation of the second heart sound with arterial hypertension of the systemic or pulmonary circuit is familiar and represents a change related to increase *in the force, or pressure, of valvular closure* (See Figure 128 for demonstration of the reversal of this effect.) Because of the double origin of P_2 the second sound in the pulmonary area may be louder than that in the aortic area in cases of systemic arterial hypertension, especially in young persons. The reason for this is that the aortic component of the second sound has been so accentuated that the combination of closure sounds is louder than the aortic closure sound alone as heard in the aortic area.

The first heart sound may be accentuated in association with hypertension of either circuit. In this situation increased force cannot be the factor in the accentuation since the valve closes when pressure in the ventricle exceeds that in the atrium and atrial pressure may not be elevated. The factor responsible for the accentuation must be increased rapidity of closure. If the phase of ventricular contraction preceding opening of the arterial valves is not prolonged then to attain the level of diastolic pressure in one or the other of the great vessels the pressure in the ventricle must rise faster than it does normally.

In so called "auscultatory alternans" which sometimes accompanies pulsus alternans (p 421) the alternately stronger and weaker heart sounds are dependent on differences in the force of valve closure.

The excursion of the valve during closure is another important factor in the intensity of the first sound at least and possibly of the second. It has already been pointed out on page 112 that the intensity of the first heart sound in various grades of atrioventricular dissociation (Fig 129) finds interpretation in the influence of atrial systole on the degree of separation of the AV valve cusps and the extent to which the belly of the cusps is displaced toward the atrium. The first heart sound is likely to be accentuated in ventricular premature contractions due to the

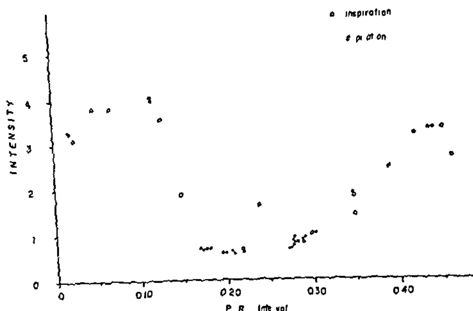


FIG. 120. The relation of intensity of the first heart sound (in arbitrary units) to the duration of the PR interval (in seconds) in a dog in which complete heart block was produced surgically by the method of Starzl and Gaertner (1434). The intensity of the first sound is presumed to be related directly to the degree of separation of the cusps at the time of contraction of the ventricle. With short PR interval one can infer wide separation of the cusps (or deep displacement of the cusp bellies) caused by atrial systole. At longer PR intervals (in the normal range) the cusps would appear to be close together, probably caused by the "breaking of the jet" phenomenon described by Henderson and Johnson (670). With still longer PR intervals there has been time for the cusps to return to a more wide open, more neutral position. Similar curves in children have been reported (1382). ("Expiration" includes readings in both the expiratory phase proper and the phase of expiratory apnea.) (Observations of Dr. S. H. Boyer IV.)

occurrence of valvular closure during early diastolic filling when the valve is relatively wide open.* The tricuspid closure sound in atrial septal defect is likely to be accentuated (see p. 349) probably because of a wide open position at the onset of ventricular systole as a result of long continuing ventricular filling. In part, the accentuated first sound in mitral stenosis may be due to displacement of the bellies of the mitral cusps far into the ventricle because of elevated pressure in the left atrium at the time that the ventricle contracts. The short excursion of the aortic leaflets in calcific aortic stenosis is a factor which tends to produce attenuation of the second sound.

* In this discussion of the relation of valve position to intensity of the heart sounds, it will be noted that the current thought is that events at the cusp belly rather than at the coapting margins are important in the production of the sound. Therefore when it is stated that the valve is wide open or almost closed it is really the position of the cusp belly that is being referred to. Movement of the margin of the cusps of the AV valves may in fact be minimal (1376).

fibrosis and calcification tend to accentuate it. With extrasystoles a faint second sound may occur largely because the cusps of the arterial valves are spread minimally. The fact that diastolic pressure is lower is also a factor, of course. With a large stroke volume it is possible that, as Rouanet predicted from his observations in a model (p. 40), accentuation of S_2 may occur from wide separation of the cusps of the arterial valves. Other clinical examples of this mechanism may be the accentuated pulmonary closure sound in atrial septal defect and the accentuated aortic closure sound in patent ductus arteriosus.

A faint first sound is a usual accompaniment of PR intervals longer than 0.20 sec., as in the first degree heart block of active rheumatic carditis (888, p. 123). The intensity of S_1 has been acclaimed as a useful clinical sign for following the progress of patients with rheumatic fever. The finding is thought to have its basis in a semiclosed position of the AV valves because of a

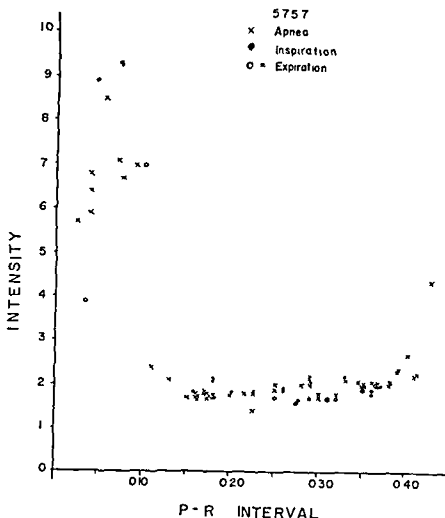


FIG 129B Relation of intensity of S_1 (in arbitrary units) to the PR interval in another dog with surgically induced complete heart block. With very short PR intervals (below 0.05 sec) and longer PR intervals (over 0.12 sec) S_1 was least loud. A secondary increase in S_1 intensity at longer PR intervals was less impressive than in the dog shown in Figure 129A. (Observations of Dr S H Boyer IV)

pulling in of the valve curtains in the wake of the atrial ejection jet.

A fourth important factor in the intensity of the heart sounds is the physical state of the cusps. This influence is seen most impressively in the instance of the accentuated sounds that occur with fibrosis of the valves. A wooden snapping or clicking sound is likely to result. The snapping character of the first heart sound in mitral stenosis probably results mainly from the fibrosis in the mitral leaflets and chordae tendineae. An early sign of rheumatic affection of the aortic valve is accentuation of the aortic second sound (Figs 130 and 131). Changes in the valve cusps are responsible for the ringing and often somewhat, although not necessarily, accentuated (intensified) "A_2" of luetic aortitis, referred to by Potain as *bruit de tabourka*. In the past (419, 739, 1112) a ringing A_2 in a normotensive individual has been accepted as *prima facie* evidence of syphilitic aortitis. The intimal atherosclerotic changes which occur secondary to the medial change and are responsible for the demonstration of calcification in the sinuses of Valsalva radiologically (874, 1055) are probably largely responsible for the change in the second sound. However, another factor which is, with that just mentioned, not mutually exclusive may be wider separation of the cusps when open because of dilatation in the region of the aortic ring (a special case of factor three above).

Ringing is a quality of the heart sounds which corresponds to musicality in murmurs. Ringing of heart sounds, as in the case of the second sound in arterial hypertension and the first sound in mitral stenosis, is represented in the spectrogram

by harmonic "knobbing" (e.g. Fig. 289). This corresponds to the harmonic banding seen in musical murmurs. A valve closure sound which is accentuated on the basis of fibrosis and calcification of the valve may have a wide frequency range with relatively little exaggeration of the intensity of the low frequency components. When the accentuation is due to increased force of closure there is an equally wide frequency range represented; however, the low frequency components are greatly exaggerated.

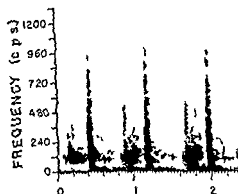


Fig. 130. Same curve A with valvular fibrosis.
Aortic area in normotensive 15 year old girl (N.B. 6,032) with early aortic valvulitis on a rheumatic basis. The second aortic sound (A) is very ringing in character with harmonic pattern and accentuation of both intensity and frequency span. These changes probably are caused by valvular fibrosis. There is an early diastolic murmur also there is probably a protosystolic click and possibly early aortic stenosis.

The sharp first sound of mitral stenosis has already been discussed. The first sound in mitral regurgitation is dull; forceful tensing of the valve is probably impossible because of the leak. In aortic and pulmonary regurgitation the second sound, even that part contributed by the valve which is diseased, is usually well preserved and often is even accentuated because of the fibrotic change in the valve and possibly because of more rapid closure of the valve. Often, the valve closure sound appears to be prolonged directly into the murmur. As can be deduced from the discussion above, an interplay of factors in aortic stenosis may result in A being increased, normal or reduced depending on which factor predominates.

In the chapter on the generation of sound in the cardiovascular system the view was ex-

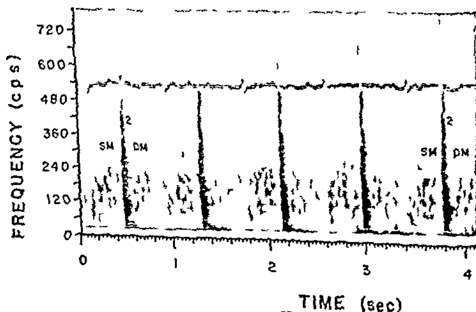


Fig. 131. Rheumatic aortic valvular disease in normotensive 20 year old patient.
There is a systolic murmur produced probably by a moderate degree of aortic stenosis. This murmur has a peak of intensity and frequency in mid systole. The second sound is followed immediately by a decrescendo diastolic murmur. The pulse pressure in this patient is not widened and the patient is in general asymptomatic. The greatly accentuated second sound (S) is noteworthy. There is a wide frequency span and intensification of the sound throughout the normal frequency range. The intensification of S_2 in this instance is caused by fibrosis of the valve. Such a fibrosed valve would be expected to produce a noisier closure.

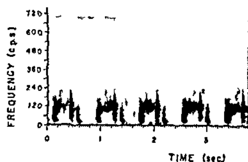


FIG 132 Protodiastolic gallop

Recorded at apex in I C (585527) 17 years old recovering from rheumatic fever He has mitral regurgitation (note the systolic murmur SM) the protodiastolic gallop (is the accentuated S_3 which usually accompanies this valve lesion

closure is brought to a more abrupt halt than normally is the case

The multiplicity of the factors influencing the intensity of the heart sounds makes it unlikely that cardiac output can be estimated thereby (472, 473, 474)

DIASTOLIC GALLOPS

Two main varieties of diastolic gallops[5] are identified (1) *protodiastolic* (ventricular, rapid filling (498) or third sound) gallop (Figs 132 and 133), which is essentially an accentuation of the normal third heart sound, and (2) *presystolic* (atrial, or fourth sound) gallop (Figs 134 135

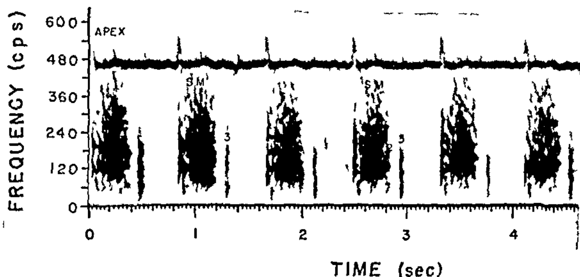


FIG 133 Protodiastolic gallop with mitral regurgitation

Apex in F I (A79811) 17 year old female who has rheumatic heart disease with mitral regurgitation producing a holosystolic murmur and accentuation of S_3

pressed that the heart sounds are valve closure sounds and that they are produced not so much by collision of the coapting margins or even by snapping of the valve curtains but rather by the generation of pressure transients when local flow responsible for valve closure is brought to an abrupt halt by the inextensible collagenous valve when its limit of stretch is attained The above analysis of factors altering the character of the heart sounds does not uncover any phenomena inconsistent with this theory In the case of the ringing sounds which accompany organic change in the valve it is possible that a very slight yielding (of which the normal valve structure is capable) is eliminated so that local flow of valve

and 136), which is essentially an accentuation of the normal fourth heart sound Both may be present in a given patient (Fig 137) Two other varieties of gallop are variations on these basic two *summation gallop* (Fig 138) occurs when both types of gallop are present and the heart rate is so rapid that they occur synchronously or in immediate succession *Mesodiastolic gallop* is the term employed when the rate is so rapid

[5] Because of the difficulties in distinguishing a gallop which has come to connote a pathologic state in all instances from its normal counterpart some such as William Evans of London (439)—have favored use of the expression *triple rhythm* This has the advantage of not prejudicing the decision of what its significance is

that the gallop sound occurs in the middle of diastole and it is impossible to tell whether it is fundamentally a protodiastolic or a presystolic or possibly a summation gallop. The term quadruple rhythm or train rhythm is used when both types of gallop are present without summation (972).

In a sense clinicians cheat a bit in deciding whether a given diastolic sound should be called a gallop or a physiologic sound. Gallops are known by the company they keep. Depending on the age of the patient and the state of the heart as evidenced by other signs, one or the other designation is assigned. Warren writes as follows (1111): "A knowledge is increased perhaps we can avoid the confusing Dr Jekyll-Mr Hyde system of nomenclature that results in naming the same sound differently as either a heart sound or gallop depending primarily on the company it keep. Actually all evidence seems to be in agreement with the statement made above that diastolic gallops are exaggerations of sounds normally present in certain individuals—indeed in the case of S_3 present in the majority of persons by graphic method when appropriate low frequency recording systems are used. The difference is a quantitative one, the normal shades imperceptibly into the pathologic. Often the line can be drawn only with difficulty. Clinical association supplemented with helpful but not conclusive tests is likely to remain the only means for drawing the line. In adults it is easier to interpret a gallop as such because of the rarity of normal third and fourth sound. Furthermore a presystolic gallop at any age can be interpreted as such since normal atrial heart sounds are not usually readily audible at any age."

The protodiastolic gallop usually occurs in conditions of diastolic overload of the ventricle either relative or absolute. For example a loud third sound gallop is characteristic of mitral regurgitation (Figs. 132 and 133), a condition in which the volume of blood entering the ventricle during diastole is increased—this volume represents something approaching a normal quota of blood plus that amount which was regurgitated during the previous ventricular systole. In this instance the ventricular overload is absolute. In myocardial disease which exclusive of mitral regurgitation is the condition in which the protodiastolic gallop is most likely to occur, the diastolic overload is relative—the normal stroke volume represents an overload for the diseased ventricle.

Protodiastolic gallops are usually loudest at the apex or left parasternal precordium but may be well heard at the sternal border. They are usually of low frequency composition. Often they are as easily felt as heard. The precordium and neck veins may show an extra impulse synchronous with the gallop sound. It is usually a good principle in listening for gallops not to try to hear an extra sound but rather to listen for the general canter rhythm that characterizes gallop. Thanks to Western cowboy cinema and television programs the rhythm of the gallop is familiar even in this modern urban era.

Presystolic gallops* are likely to be loudest in the vicinity of the sternum. They may be loud even in the aortic area. Again they are of low frequency content although in general perhaps more snappy than are protodiastolic gallops.

The presystolic gallop seems to occur mainly with conditions of systolic overload of the ventricle: systemic arterial hypertension (Fig. 134), aortic stenosis (Fig. 135), pulmonary arterial hypertension, pulmonary stenosis (Fig. 136). This association is the basis for the fact that the presystolic gallop was termed bruit de Bright (from its occurrence with Bright's disease) by French authors of the last century and Nieren galopp (kidney gallop) by the Germans during the

*Lian (917, 931) speaks of "galop présystolique retardé." With his phonocardiograph which is "ear like in its frequency response," he records vibrations preceding the summit of the R wave of the electrocardiogram in some patients and not in normal persons. Leonard and co-workers (836) find that the ordinary presystolic gallop can be caused to move toward and into the first part of the first heart sound by the application of tourniquets to the extremities. These observations indicate that the initial vibrations of S_1 may at times be indeed of atrial origin although their persistence in atrial fibrillation suggests that in some cases at least they have other basis. Duchosal (36) concluded that the interval between the presystolic gallop and the first sound is an indication of the severity of the cardiovascular derangement. When a patient with a presystolic gallop improved, the gallop moved progressively into the first sound.

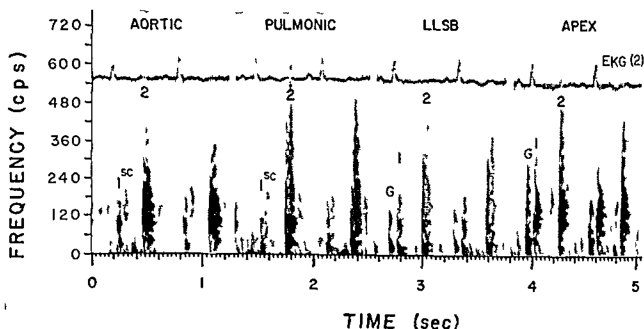


Fig 134 Presystolic gallop

Recorded at indicated areas in W B (593724) 40 year old patient with malignant hypertension At the base *there is a protosystolic click (c) which is* undoubtedly of aortic origin At LLSB and the apex the presystolic gallop (G) is striking

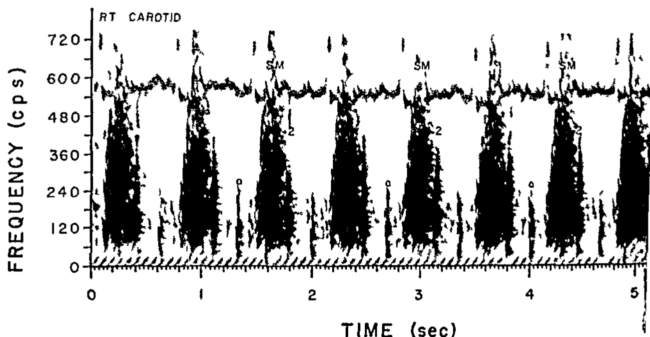


Fig 135 Calcific aortic stenosis with atrial gallop recorded in the neck

1 A M D (411599) 73 year old white female has calcific aortic valve disease BP is 206/76 There was no definite aortic diastolic murmur The pulses were of water hammer type and there were capillary pulse She was anemic with hematocrit of 30 per cent This and arteriosclerosis may account for the wide pulse pressure

The right carotid record shows the transmission to the neck of the non y murmur and an atrial sound Precordial records showed the musical murmur characteristic of the Gallavardin phenomenon (p 267)

That the atrial gallop is right sided in origin because it was audible almost exclusively in the neck could not be concluded with certainty Possibly some left atrial gallops are also more likely to be audible in the neck than at the precordium (See fig 135B)

same period Perhap surprisingly, Weitzman (1573) failed to find atrial gallop with increased frequency in aortic valve disease The remainder of his experience is consistent with mine he found it frequently in hypertension but only when ventricular hypertrophy was present However of those patients with the sign 77 per cent were free of congestive failure and 25 per cent were totally asymptomatic When the PR interval is prolonged an atrial gallop is especially likely to occur (523) or at least is more readily audible (see below)

Because the second sound is often accentuated in the cases in which presystolic gallop occurs and because the presystolic gallop is likely to be striking at the base the rhythm of the presystolic gallop is often anapestic (1191) in terms of meter short short long ⌣ ⌣ —, the accent being on the second heart sound Some have considered it a helpful mnemonic trick to refer to the presystolic gallop as a Tennessee gallop and to the protodiastolic gallop as a Kentucky gallop the accent being on the second heart sound in each case It is only fair to point out, however, that the device is often more confusing than helpful In some areas of the precordium either type of gallop may resemble either Tennessee or Kentucky This device is possibly as useless as the proposal made in 1881 by Fraentzel (477) to classify gallops as hunting, ordinary, or school depending on the type of gait most resembled (709) Although one would scarcely recommend it seriously as a mnemonic device Morse code for *u* (—) and for *f* (—) would be as valid as some of the tricks suggested

Because of the difference in the clinical setting in which the two types of gallop occur the prognostic significance is different Excluding the protodiastolic gallop of mitral regurgitation protodiastolic gallops have more grave prognostic connotations than do presystolic gallop On an average in adults the life expectancy with a protodiastolic gallop is three to four years (654) which although longer than previously thought is a grave prognosis

With carotid sinus pressure (588) one may because of the slowing of heart rate and prolongation of diastole be able to determine whether a gallop which before could only be called meso diastolic is presystolic or protodiastolic, or if a quadruple rhythm becomes evident one can conclude that the gallop was of the summation type With carotid sinus pressure a protodiastolic gallop may disappear through improvement of myocardial competence at the slower rate (1250) Laubry and Harvier (849) used the bradycardiac effect of ocular pressure in the study of gallops

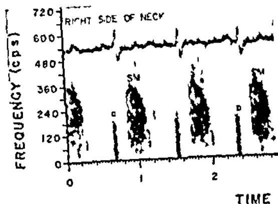


FIG 13aB A very similar finding (see fig 13A) in L C (1955122) age 7 who has little evidence of obstruction at the aortic valve but has an exceedingly loud musical aortic systolic murmur At the base of the neck there is a presystolic gallop

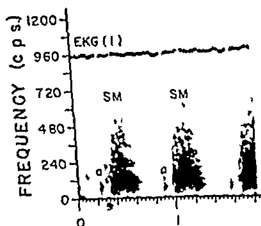


FIG 13C Presystolic gallop (a) in association with pure pulmonary stenosis Note Christmas tree configuration of the systolic murmur and the absence of a gap between the end of the murmur and the aortic component of the second sound (which is buried in the end of the murmur)

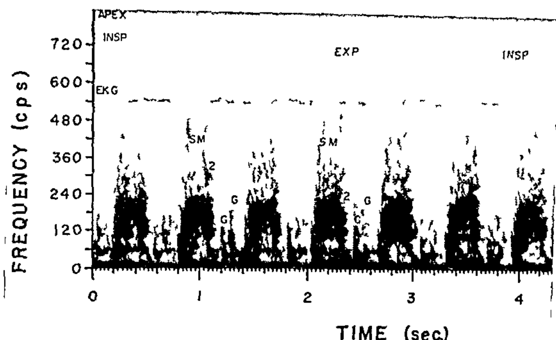


Fig. 137 Double gallops

Both protodiastolic and presystolic gallops in T. S. (670561) 35 year old female with hypertension, posterior myocardial infarction, probable multiple pulmonary emboli and congestive heart failure. The P-R interval measured 0.23 sec. Note that the systolic murmur has a tendency to musicality.

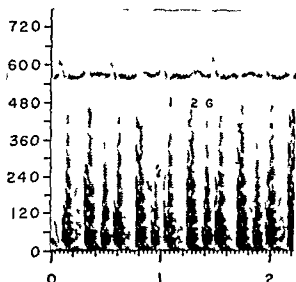


Fig. 138 *Mesodiastolic gallop*, probably summation type. *The patient (C. C. 196902) had severe systemic* arterial hypertension.

Carlgren (235) studied gallops with the multiple filter system of Mannheimer ("calibrated phonocardiography") and concluded that gallops have a predominant frequency in excess of 100 c.p.s. as compared *with the normal counterparts which* tend to lie below this level of frequency. Although possibly statistically true, the distinction *is not great* enough to permit differentiation of *gallops and normal sounds in the individual case.* Furthermore, Frost (497, 498) could not corroborate Carlgren's findings.

Dock (358) has described a *presystolic sound* gallop if you will, over the jugular veins associated with *high* venous pressure. *This* sound appears to be caused by a centrifugal wave from the atrium when there is sinus rhythm and either *tricuspid stenosis or elevated end-diastolic pressure* in the right ventricle. Often a presystolic gallop was not demonstrable over the precordium. Usually a giant *a* wave was present in the neck veins with the sound near its peak. The sound *occurred 0.10 to 0.16 sec. after the onset of the* P wave of the EKG. Our experience would not indicate that the sound recorded by Dock is specific to the conditions he mentioned or of particular diagnostic utility. Groedel and Miller *(604) found the sound frequently and in many* different conditions. We have found it for example in calcific aortic stenosis, possibly merely because auscultation in the neck is more regularly practiced in these patients who in addition are *prone to have a presystolic gallop* (see Fig. 135 A and B).

The atrial wave of the apex cardiogram is of large size in cases of presystolic gallop (128, 876).

This may support the view that the presystolic gallop is a vibration generated in the ventricle as a result of atrial systole.

Duchosal (377) enunciated two laws with reference to the PG interval (the interval between the P wave of the EKG and the atrial gallop):

"1. The duration of the PG interval is an index of the seriousness of the affection.

"2. The PG interval varies in proportion to the amelioration or the aggravation of the case during its evolution."

Duchosal stated further: "We are of the opinion that the variations in the PG interval arise from the variations in the force and volume of the auricular wave, while the hypotonicity regulates the production and the acoustic quality of the sound. Muscular hypotonicity of the ventricle is necessary for the production of gallop rhythm."

Leonard and colleagues (876) have provided convincing confirmation for Duchosal's laws by demonstrating that the PG interval can be reduced by impounding blood in the extremities with tourniquet.

Duchosal (376) points out that the audibility of a presystolic gallop is dependent on at least four factors: (1) The duration of the gallop sound, (2) the PP interval, (3) the PG interval, and (4) the R-S_1 (Q-1) interval. Leonard *et al.* (875) demonstrate *that* S_1 *is often delayed in hypertension* and suggest that increased *audibility of a presystolic gallop in hypertension can be attributed in part to this fact.*

At various times a biventricular *gallop*, that is a gallop sound produced in each ventricle which in the composite may resemble a short *murmur*, has been suggested (536). Although a distinct possibility, such a double gallop awaits more detailed study for confirmation.

The gallop is both one of the most important and one of the most difficult of the auscultatory phenomena. Some clinicians consider it adequate basis for digitalization; they even question an impression of heart failure if a gallop is not present, at least not present after light exercise. Some would not diagnose gallop if the rate is not in excess of 90, certainly 100. Others would consider as a gallop any exaggeration of a normal diastolic sound. Such exaggerations can occur at slow rates and are in fact more significant under these circumstances.

THE EARLY DIASTOLIC SOUND IN PERICARDIAL DISEASE. In constrictive pericarditis a sound in early diastole (see pp. 127 and 419) may be very loud, even louder than either the first or the second sound. Probably this sound is of the same nature as the protodiastolic gallop. Its character, such as the close position to the second sound, is probably determined by the physiologic conditions peculiar to this disease, e.g., the high venous pressure and early arrest in diastolic filling. In pericardial effusion there occurs a protodiastolic gallop indistinguishable from that of myocardial disease (1078) or with timing and other characteristics intermediate between those of ordinary protodiastolic gallop and the sound of constrictive pericarditis.

The protodiastolic sound of constrictive pericarditis is more likely to occur *when the pericardial scar is calcified.* It may, in combination with the second sound, simulate a split second sound.

SYSTOLIC CLICKS

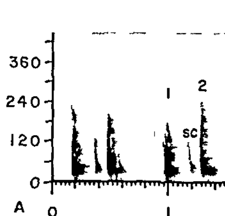
Clicks may be early systolic, midsystolic or late systolic. They may be single or multiple. They can be further classified on the basis of their seeming mechanism of genesis (see p. 129).

1. *Clicks caused by pleuropericardial adhesion* (usually late, occasionally mid, rarely (686) protosystolic). (See Figs. 139 to 144.)

2. *Clicks caused by costochondral or chondrosternal motion* (usually midsystolic). (See Figs. 145 to 149.)

3. Clicks caused by dilatation of hypertension and other diseases of (a) aorta, (b) pulmonary artery (always early systolic). (See Figs. 151 to 154.)

4. Miscellaneous: (a) Atrial sound in systole caused by low nodal pacemaker with retrograde conduction ("ectopic atrial gallop") (276, 686), complete heart block (538), atrial flutter (Fig. 443); (b) mediastinal emphysema; (c) left pneumothorax; (d) plaques of calcification in the pericardium (see Fig. 429); (e) caused by impact of expanding ventricular aneurysm (Fig. 156) or auricular appendage (Fig. 155) on pericardial structure (early or midsystolic).



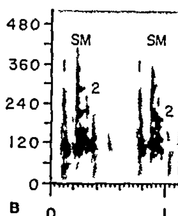


FIG 139 FIG 140

FIG 139 Spectrogram of *midsystolic click* presumably caused by pericardial adhesion FIG 140 Same introducing systolic murmur which is thought to be extracardiac in origin probably caused by pericardial roughening



FIG 141A *Midsystolic click with fibrothorax*

N S (153008) 53 year old male had pulmonary tuberculosis resulting in left sided fibrothorax which produced an immobilized retracted left hemithorax and drew the heart to the left The SPCG shows a mid systolic click (see Fig 141B)

Among all cases of gallop rhythm studied by Thompson and Levine (1472), 16 per cent had an extra sound in systole responsible for the canter Although this provides some notion of the relative frequency of systolic clicks it must be noted that early systolic clicks and probably some late systolic clicks would not be counted in a listing of cases of gallop rhythm

The quality of systolic clicks was well described by Potain (1224), who referred to them as "small, sharp, clicking sounds, well localized and such that one can scarcely attribute them to anything except the tensing of a pericardial adhesion"

In the spectral phonocardiogram (see the many accompanying illustrations) systolic clicks are characterized by (1) short duration (2) relatively pure frequency content and (3) the fact that the "frequency bottom" usually is not at zero" These spectrographic characteristics are shared with the mitral and tricuspid opening snap (p 191 *ff*) and with the pulmonary reversal snap of pulmonary stenosis (p 374) Do the valve closure sounds truly have components down to the frequency of a few cycles per second? How many cycles or what fraction of a cycle is necessary to activate the display mechanism of the spectrograph? If one complete cycle is necessary for example then a 25 c p s vibration would need to be at least 0 04 sec in duration to be represented Clicks and snaps may be too brief for their low frequency components to be demonstrated On the other hand because the valve closure sounds are commonly palpable it is plausible that they should be found to have subaudible frequency components It is likewise plausible that snaps should have relatively pure frequency composition well up in the audible range In brief it is likely that snaps have little or no subaudible frequency composition however even if subaudible components of very low intensity were present, the brief duration of

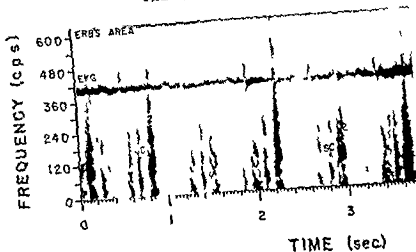


Fig 141B (See Fig 141A)

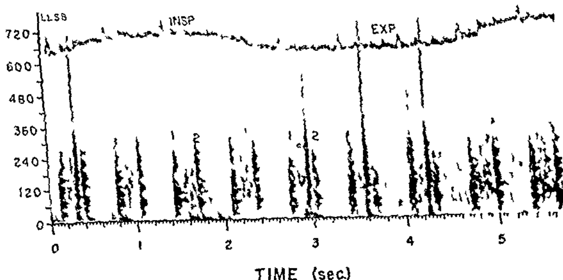


Fig 14[illegible] Late systolic click only in expiration LLSB Bx *not clear* H W (731037) had healed bacterial endocarditis of the aortic valve

the sound would preclude demonstration in the spectrogram as now made

The clicks due to pleuropericardial *adhesions* are usually loudest at the apex or the lower left sternal border This type of click constitutes the *bruit de triolet* of Cuffer and Sarda and is the *claquement mésosystolique pleuro pericardiaque* of Lian (920) These clicks may with the second sound produce a combination which can be confused for a widely split second heart sound *or* a second heart sound plus mitral opening snap The latter bit of mimicry may of course suggest mitral stenosis (917) Occasionally as one would anticipate patients with well *confirmed mitral* stenosis (e g M W 726338) have a late systolic click in addition to the signs of mitral stenosis Clicks of this variety are *frequently* encountered as a more or less permanent *residuum of* acute pericarditis of rheumatic (1o8) *or* idiopathic origin With respiration or position of the patient there may be variation both in the intensity and timing (7 0) of the click (Figs 142 and 143) Sometimes a short murmur is initiated by a *click of this* type *and* sometimes the murmur is musical (see p 207)

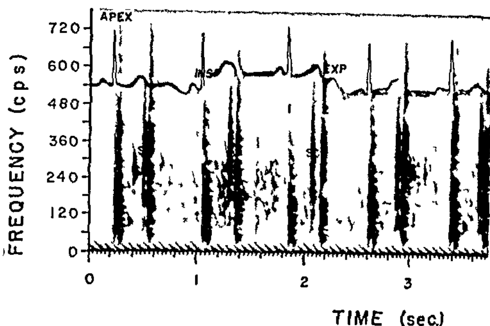


Fig 143 Late systolic clicks varying in timing with respiration

Incidental finding in T R (174366) 39 year old female with a normal heart This recording at the apex shows that with inspiration the click occurs earlier in systole

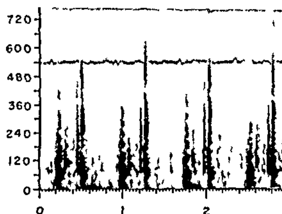


Fig 144 Early and late systolic clicks

Recorded in a 58 year old woman (688712) who had ARF in youth and displayed mild trichterbrust

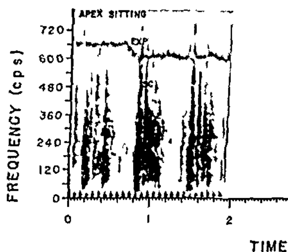


Fig 145 Multiple clicks with the Marfan syndrome

D D (758872) age 16 years has slight dislocation of the lenses and a habitus compatible with the Marfan syndrome Recording at LLSB with patient sitting At the apex although the main clicks were in systole as they were at LLSB there were also less conspicuous ones in diastole

This type of murmur which is introduced by a *systolic click whether noisy or musical is thought* to be produced by rubbing of pericardial surfaces Clicks of probable pleuropericardial origin may occur in early diastole in patients who also have click(s) in systole (76A)

Systolic clicks *due to costochondral or chondrosternal* motion are heard in three types of situation, occurring singly or in combination First they occur in persons with pectus excavatum pectus carinatum ("pigeon breast") and other types of chest *deformity* in which even the normal heart is likely to impinge on the thoracic cage

In the Marfan syndrome (Figs 145, 146 and 147) *such clicks are particularly frequent* in part because of the occurrence of these chest deformities in part because of the loose jointedness which extends to thoracic structures and of course at times because of enlargement of the heart *Secondly* they occur in patients with normal chest walls and normal hearts but a heart

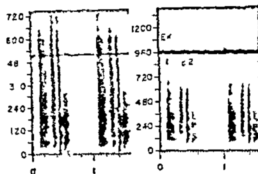


Fig 146 Multiple systolic clicks with the Marfan syndrome

Apex in M B (697662) 46 year-old woman with Marfan's syndrome aortic regurgitation left ventricular enlargement The multiple systolic clicks were described by the stethoscopist as a systolic crunch Because of the mild chest deformity cardiac enlargement and general looseness of joints associated with the connective tissue disorder the systolic clicks are thought to have resulted from movement of joints of the bony thorax

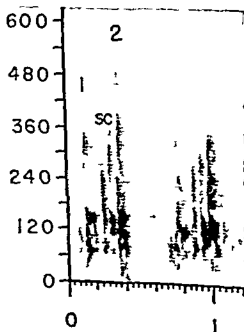


Fig 147 Pulmonary area in patient (M B 673169) with massive ascites caused by intra abdominal malignancy heart presumably normal The *multiple systolic clicks* probably are caused by movement of costochondral and chondrosternal joints by the displaced heart

displaced for some reason such as massive ascites (Fig 147) or pregnancy Thirdly they occur in patient with a normal thoracic cage but an enlarged heart

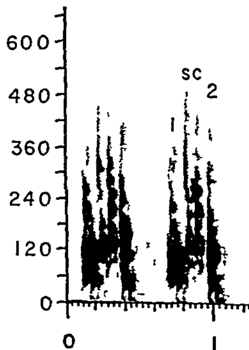


Fig 148 Typical *xiphosternal crunch* at LLSB in O F (375948) Confusion for a pericardial friction rub occurred when the patient was admitted with fever of uncertain origin

It is possible that in rare instances a click in early diastole is of costochondral and or chondrosternal origin (Fig 149) This category includes the xiphosternal crunch (Fig 148)

An early systolic click occurs at the base of the heart in association with disease of the great vessel In dilatation and/or atherosclerosis of the ascending aorta especially with systemic arterial hypertension an early systolic click is heard in the aortic area (Fig 150 to 152) In dilatation of the pulmonary artery (Fig 153 and 154) especially with pulmonary hypertension an early systolic click (called pulmonary ejection sound by Leatham and Vogelpoel (866)) is a very frequent and often very conspicuous finding The proximity of the pulmonary artery to the anterior chest wall may account for this phenomenon's being a more striking feature with disease of the pulmonary artery than with corresponding disease of the aorta The early systolic click is loudest in expiration It may give an impression of an unusually clicky first heart sound since the first sound itself may be inconspicuous at the base

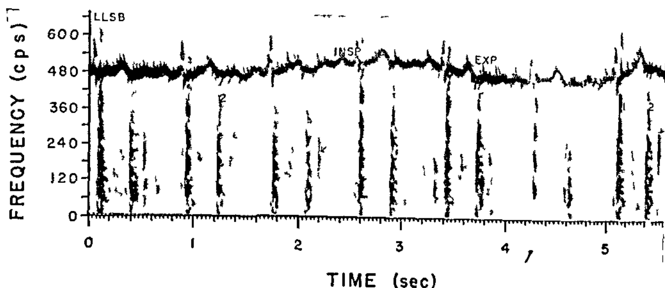


Fig 149 Early diastolic click of extracardiac origin

H H (763162) 41 year old female had a typical xiphosternal crunch displayed better in records from other areas Of particular interest in this recording at LLSB is the early diastolic click *Diastolic clicks of extracardiac origin* are demonstrated also in Fig 145

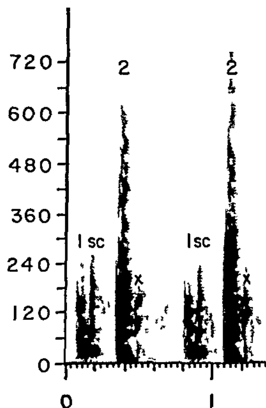


Fig 150 Early systolic click

Aortic area in 33 year old patient (A W 672911) with systemic arterial hypertension with mild dilatation of the aorta and kinked carotid A_2 is accentuated in intensity and frequency span There is a striking protosystolic snap followed by a short murmur The sound in early diastole (X) is of uncertain origin It is probably too close to A_2 to be a protodiastolic gallop A similar early diastolic sound may be demonstrated in Figure 134

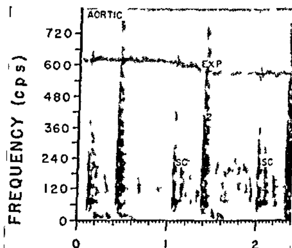


Fig 151 Early systolic click with the Marfan syndrome

W M (702409) 27 year old male has well established Marfan syndrome and is a member of a family with several affected members A faint aortic diastolic murmur at Erb s area with some enlargement of the left ventricle is present but the aorta is not dilated by x ray and the patient is asymptomatic The record here from aortic area shows an early systolic click which becomes most inconspicuous in inspiration It is probably related to the dilatation of the base of the aorta which is also responsible for the aortic regurgitation

Even in phonocardiograms the early systolic click can masquerade as the first sound The *abnormally great distance between the beginning* of the QRS and the extra sound is a clue that it is not S_1 but when there is a question of the presence

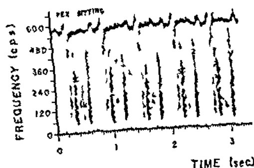


Fig 152 Early systolic click in patient with mild aortic stenosis and aorta bound in fabric scultetus binder after resection of aneurysm caused by cystic medial necrosis (1081) Apex in A. H. (14094) who (18 months previously) had the operation described

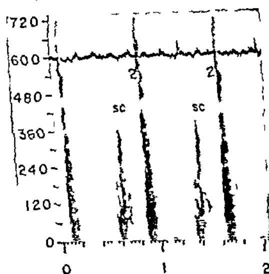


Fig 153 Early systolic click

Pulmonary area in patient with pulmonary hypertension and dilated pulmonary artery The early systolic click initiates a faint systolic murmur An even fainter early diastolic murmur immediately follows the accentuated 2

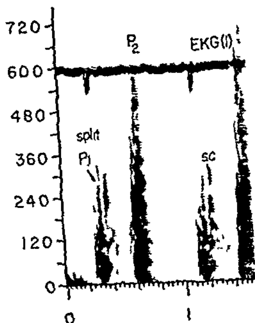


Fig 154 Early systolic click

Pulmonary area in patient (Z. O. 673149) with atrial septal defect and pulmonary hypertension. P_2 is accentuated and split the second component (pulmonary closure sound) being the louder. P_1 is split and is followed by a protosystolic click. There is a triple complex in the vicinity of S_1. The patient subsequently was found to have an atrial septal defect of the ostium primum type with no abnormality of the AV valve

of mitral stenosis it may be difficult to be certain whether the sound is a delayed mitral closure sound or a pulmonary early systolic click. In the SICC the frequency characteristic (see above) permit differentiation in the majority of instances. When the first sound is present followed closely by the systolic click an impression of split first heart sound is likely to be created. For example others (888 Fig 34) present what they interpret as a widely split S_1 in atrial septal defect it is rather clearly S_1 followed by an early systolic click

In addition to dilatation of and hypertension in a great vessel increased volume of flow seems clinically to be a feature of cases in which an impressive early systolic click occurs. For example the pulmonary click is particularly striking in cases of atrial septal defect (Fig 154) and left-to-right shunt despite little elevation of pulmonary pressure. Furthermore in tetralogy of Fallot especially with pulmonary atresia or severe pulmonary stenosis so-called pseudotruncus arteriosus in which most of the blood is passing out the dextroposed aorta there is likely to be a click over the aorta heard best at the left of the sternum (975) After Blalock Taussig or Potts operation the early systolic click tends to be especially pronounced

Dock (357) and Leatham (862) state that the early systolic click occurs later if diastolic pressure *is very high* in the pulmonary artery. The interval between the onset of the QRS (or S_1) and the click

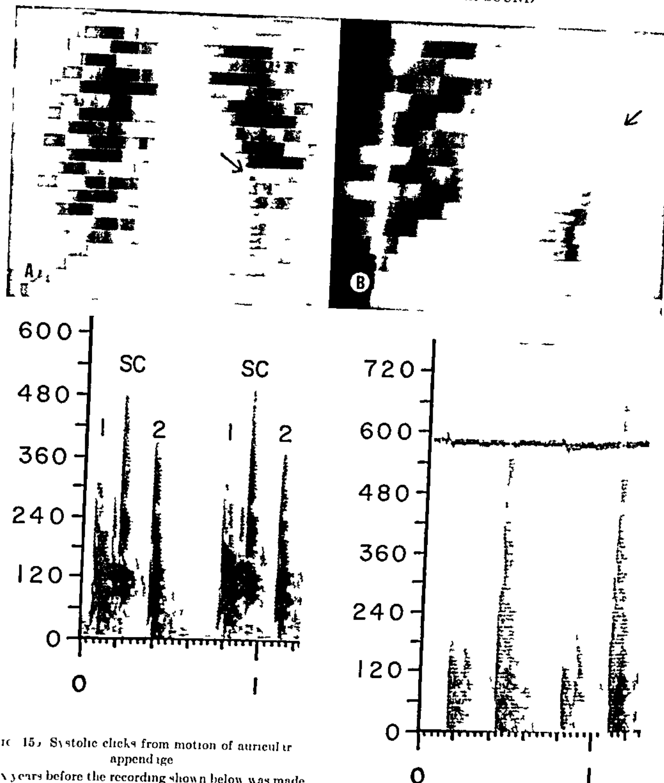


Fig. 155 Systolic clicks from motion of auricular appendage

Six years before the recording shown below was made the patient (F. H. 452300) had staphylococcal endocarditis on the mitral valve previously damaged it was thought by rheumatic fever. On fluoroscopy and roentgen kymography (postero anterior (A) and LAO (*B*)) the left auricular appendage uncoiled rose out of its bed in the AV groove and probably expanded as did the rest of the atrium. The arrows indicate the areas of left atrial outpouching synchronous with ventricular systole as indicated by inward movement of the ventricular border. The peculiar movement was probably responsible for the peculiar sound.

Fig. 156 Early systolic click with ventricular aneurysm

LLSB in W. D. (684406) 50 year old with old anterolateral myocardial infarction and ventricular aneurysm at lower left heart border. Expansion of the aneurysm in early systole is possibly the mechanism of the sound. In spite of the loud second sound the patient was normotensive; the finding may be the result of valvular sclerosis. Furthermore it is also possible that the early systolic click is of aortic origin and not produced at the ventricular aneurysm.

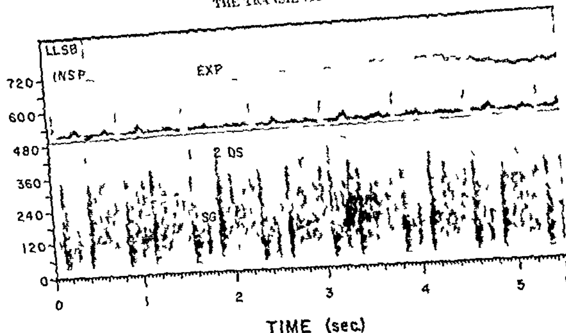


Fig 15 Systolic gallop in aortic regurgitation

LLSB in O M (704573) 24 year old patient with a history of acute rheumatic fever at the age of ten years There were peripheral signs of marked aortic regurgitation The mid systolic sound is more thudding than the usual systolic click There is a loud decrescendo diastolic murmur of aortic regurgitation

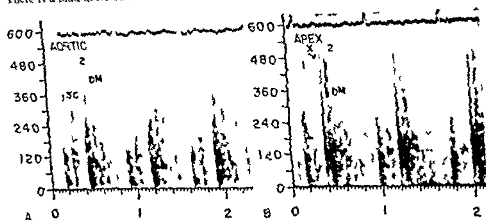
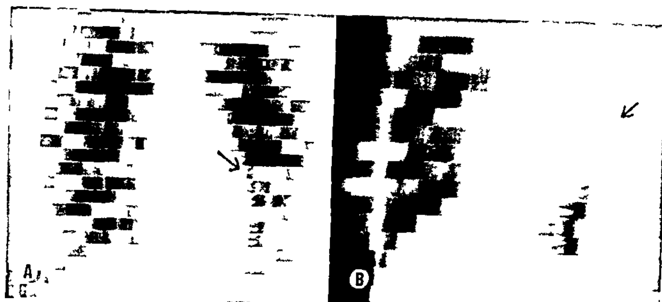


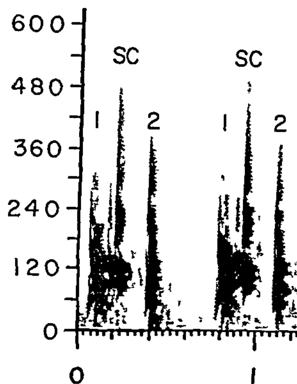
Fig 15a Circumscribed systolic sound in syphilitic aortitis with aortic regurgitation

In the aortic area (A) of this patient there is demonstrated a decrescendo murmur in diastole beginning immediately with S_2 Early in systole there is a circumscribed sound marked SC (systolic click) which is not actually a click as far as either its spectral pattern or the impression it made on the ear are concerned To the ear there appeared to be wide splitting of the first sound

At the apex (B) of the same patient there is a late systolic click (X) In the spectrogram this is clearly a click because of its homogeneity of intensity frequency pattern its brevity and the fact that its frequency bottom does not quite reach the base line as is the case with the valve closure sounds To the ear this sound created the impression of a split second sound A pericardial origin is suggested

is an index of isometric contraction which may be prolonged with pulmonary hypertension The interval is usually of the order of 0 07 sec In mild or moderately severe cases of pulmonary stenosis an early systolic click occurs but is earlier than in cases of pulmonary hypertension and earlier than the early systolic click of dilated aorta (866)





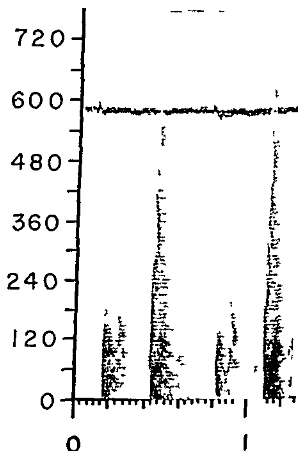


FIG 155 Systolic clicks from motion of auricular appendage

Six years before the recording shown below was made the patient (F H 452300) had staphylococcal endocarditis on the mitral valve previously damaged it was thought by rheumatic fever On fluoroscopy and roentgen kymography (postero anterior (A) and LAO (B)) the left auricular appendage uncoiled rose out of its bed in the AV groove and probably expanded as did the rest of the atrium The arrows indicate the areas of left atrial outpouching synchronous with ventricular systole as indicated by inward movement of the ventricular border The peculiar movement was probably responsible for the peculiar sound

FIG 156 Early systolic click with ventricular aneurysm

LLSB in W D (684106) 50 year old with old anterolateral myocardial infarction and ventricular aneurysm at lower left heart border Expansion of the aneurysm in early systole is possibly the mechanism of the sound In spite of the loud second sound the patient was normotensive the finding may be the result of valvular sclerosis Furthermore it is also possible that the early systolic click is of aortic origin and not produced at the ventricular aneurysm

con i t of at le t two closely timed click The mechanism was thought to be impact of the auricular appendage on the pericardium In 1947 when the patient was 14 years old he had staphylococcal endocarditis of the mitral valve which was cured with penicillin While he was still convalescing it was noted on fluoroscopy that there was a bizarre outpouching of the left auricular appendage with each ventricular systole This has persisted throughout the last t e e r s during which time the patient has borne four children and displayed no symptoms or evidence of cardiac enlargement The outpouching has been timed and documented by means of roentgenkymogram (Fig 1) A and B) and cinefluorogram Temporally the systolic click is found to correspond precisely with the unfurling of the auricular appendage The systolic click is loudest in the left midprecordium somewhat removed from the apex a fact which may account for the lack of much systolic murmur of mitral regurgitation in the record of Figure 1 C

There is a question whether there is anything legitimately termed systolic gallop Systolic clicks may result in a combination with a e nt r rhythm when tachycardia is for some reason present I have avoided the term systolic gallop because of the benign nature of what we instead term systolic clicks and the lack of the relatively grave prognosis associated with diastolic gallops Most systolic clicks are just that and by their clicking nature usually differ in quality from gallop sound Wolferth (1o7) believes that aside from these true clicks there are occasional more *thudding sound* in systole to which the designation of gallop can be assigned with validity In 1940 with Margolies (1o79) he described the mid

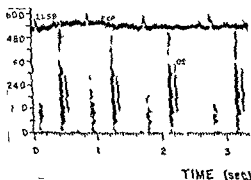


Fig 160B (For comparison with Fig 160 A)—a typical mitral opening snap of mitral stenosis From the left lower sternal border in W S G (348 7)

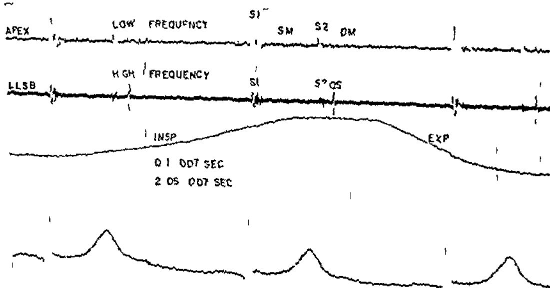


Fig 161 Opening snap of mitral stenosis

Apex and LLSB in F T (23013) 37 year old female High frequency recordings display opening snaps especially well whereas low frequency recordings are better for demonstrating the diastolic rumble of mitral stenosis It was for this reason that the recordings from the areas most likely to show one or the other of these phenomena were made with the filter system indicated The Q 1 and 2 OS measurements (see p 2) are indicated

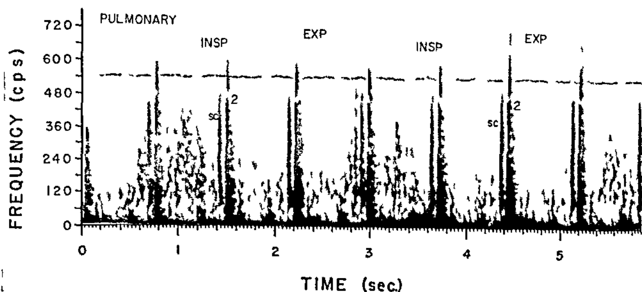


Fig. 159 The *late systolic click* demonstrated here was loud over the entire precordium in M. C. (A153?5) 15 year old female who was observed during an attack of acute rheumatic fever several months previously. During the peak of the attack a pericardial friction rub was present.

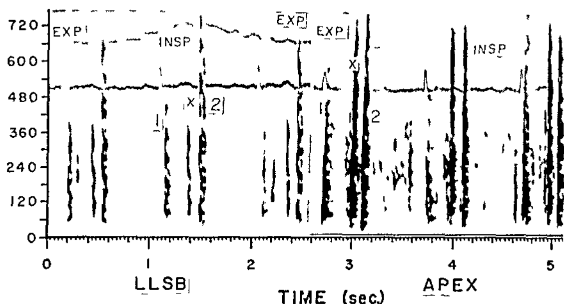


Fig. 160 Late systolic click simulating mitral stenosis.

LLSB and apex in D. P. (B. C. H. 211864) 13 year old female. (Photographic record.) Mitral stenosis was suspected *because of what was stethoscopically interpreted as S_1 followed by an opening snap.* The late systolic click is labelled X. The display of the recording from the apex was made with greater amplification so that background noise is more evident and the sounds in general appear louder.

Rarely, systolic clicks may be associated with post infarctional aneurysm of the ventricle (Fig. 156). Although *it is possible that pericardial* adhesion is the mechanism it is also conceivable that the impact of the expanding aneurysm on surrounding structures is responsible. The click described by Leroy and Roberts (882) and by Frost (498) was mid systolic in timing. I have observed an early systolic click in a patient with an aneurysm origin in the aorta in this atherosclerotic individual cannot be excluded. Mandims (1030) describes a similar case.

I have observed a case unique in my experience and possibly in the literature in which mitral regurgitation *produced systolic expansion of the* left atrium, with uncoiling of the auricular appendage and was associated with a systolic click which graphically (Fig. 155) was seen *to*

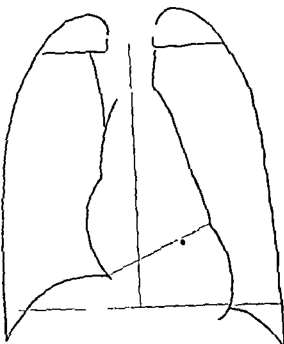


Fig. 164 The area of maximum audibility of the opening snap in 15 cases. The large dot indicates the position in the patient from whom the orthodiagram shown here was made. Differences in heart size and shape were not controlled. The lowest circle for example was in a patient with a very large heart. Compare the distribution of the dots with the location of the mitral ring as indicated by calcification of the annulus fibrosus mitralis (Fig. 319). (Courtesy of Margolies and Wolferth (1040) and *American Heart Journal*.)



Fig. 165 Predominant mitral stenosis with dense calcification (arrows) in mitral valve. Compare with Margolies and Wolferth's map of the opening snap (Fig. 164). (Courtesy of Cosby and Sloan (291).)

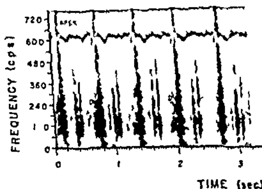


Fig. 166 Opening snap as first clue of mitral stenosis

R. S. (F.1932) 50-year-old male has had severe bronchial asthma all his life and shows signs of advanced pulmonary emphysema. An earlier record showed an opening snap as the first suggestion of mitral stenosis. At that time he had a respiratory infection with right-sided heart failure. With clearing of his infection, heart failure and bronchospasm the classical auscultatory signs of mitral stenosis appeared (*see above*). The signs were atypical only in that the presystolic murmur was heard amazingly low—at the left costal margin just inside the midclavicular line. Late in systole just before S_1 there is an extracardiac click.

opening snap (Fig. 160). Finally, the identification of a systolic click in certain areas of the precordium or with certain positions of the patient may suggest the extracardiac and therefore innocent nature of a systolic murmur.

ATRIOVENTRICULAR OPENING SNAPS

The best studied is the mitral opening snap of mitral stenosis (Fig. 161). Relatively recently (816) the tricuspid opening snap has been characterized.

In mitral stenosis the opening snap occurs shortly after the second heart sound. The length of the interval between it and the opening snap is determined mainly by the level of left atrial pressure or better by the differential pressure or pressure gradient between left atrium and left ventricle at the time of closure of the aortic valve (Fig. 162). (Wells (1527) thinks the degree of fibrosis in the valve also influences the interval (see p. 292).) Systemic hypertension is accompanied by a longer S_2-OS interval than would be the case with the same degree of mitral stenosis and no hypertension. The same effect is produced

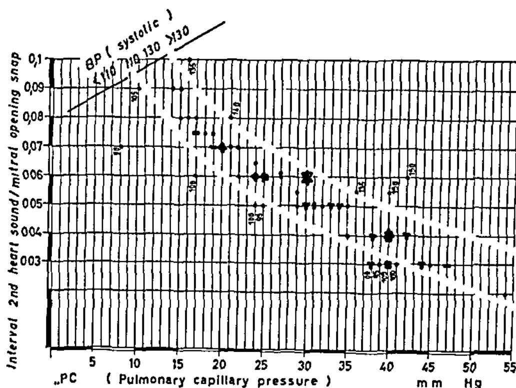


Fig. 162. Relationship between S_2 OS interval and pulmonary capillary pressure in mitral stenosis. The figures indicate systolic arterial pressure determined by means of the arm cuff. There is a close depend[…] between the two parameters. With elevated systemic pressure S_2 OS is relatively long; with low systemic pre[…] it is relatively short. (From Wolter *et al.* (1585).)

systolic sound of aortic regurgitation, referring to it as a "systolic gallop." This usage seems entirely legitimate because consistent with its more "thudding" quality, and the SPCG (Figs. 157 and 158) shows that the sound extends to the frequency base line as do gallops and valve closure sounds. Its location in mid systole makes it unlikely that the mechanism is the same as that of the early systolic click of aortic or pulmonary artery origin. The explanation of Wolferth and Margolies (1579)—that it is due either to sudden checking of aortic distension or to impact of the aorta on surrounding structures—seems entirely plausible. Johnston (751) concluded that whereas pleuropericardial mid systolic clicks occur most often at the apex, the thudding mid systolic sound to which the term systolic gallop is applicable occurs most often in the aortic area. Gmachl (558) also describes a distinctive mid systolic gallop of aortic regurgitation.

The systolic click is usually of benign significance—the early systolic click is an exception. The main practical clinical reason for a familiarity with them is that grave conditions may be simulated. The systolic click, especially with tachy-

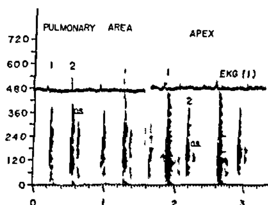


Fig. 163. Mitral stenosis.

Recorded at areas indicated in case with all hallma[…] of mitral stenosis except unequivocal rumble. N[…] broad notched P wave of EKG; opening snap louder [in] pulmonary area; apical first sound (1) accentuated [in] both intensity and peak frequency and delayed in re[…]tion to QRS.

cardia, may create a canter suggesting diasto[…] gallop. The two may be very difficult to di[…]guish, with the precise timing of the extra sou[…] determined only by phonocardiography. The l[…] systolic click may, with the second sound, pr[…]duce a combination suggesting second sound [.…]

by aortic stenosis and possibly by aortic regurgitation. The interval is shortened immediately after exercise because of rise in left atrial pressure and is prolonged with administration of norepinephrine because of the elevation of systolic pressure in the left ventricle (64). The Valsalva maneuver is likely to result in prolongation of the S₂-OS interval. The S₂-OS interval is variable in atrial fibrillation, being longest after longer preceding diastolic periods (Fig. 163).

The mitral opening snap is best heard in the left midprecordium but may be quite distinct in the aortic area, and rather as a rule is louder at the left sternal border than at the apex (Fig. 163). A scatter diagram of the point of maximum audibility on a drawing of the rib cage (1040) tends to show (Fig. 164) a concentration in the area where calcification in the mitral valve is visualized on postero-anterior chest x-ray (Fig. 165).

In combination with the second sound the opening snap suggests a widely split second heart sound. The fact that the opening snap is often loud at the base contributes to this confusion. This confusion is probably the basis for the mistaken impression that a split second sound is characteristic of pulmonary hypertension in general and of mitral stenosis in particular. The mitral opening snap usually has a drier—indeed a snappier—quality than do valve closure sounds; however, by no means is it always easy to differentiate it by ear from a split second sound. The spectral phonocardiographic features assist the identification.

Evan (914) emphasizes that auscultation in the suprasternal notch assists the differentiation between a split second sound and a second sound followed by opening snap. A mitral opening snap is usually well heard in this area. A split second sound usually is not, whilst second sound is heard in the suprasternal notch largely of aortic valve origin.

In the SLCC the opening snap has the same characteristics as systolic click (see p. 180): (1) short duration; (2) tendency to pure frequency content; (3) failure of the 'frequency bottom' to reach zero. The differentiation from the second part of a split second sound is clinched by the demonstration of two separate distinct components of the second sound preceding the opening snap. This is of course most likely to be possible with inspiration and in recordings from the pulmonary area, where, fortunate for this observation, the opening snap is usually well heard and demonstrated.

A prominent mitral opening snap indicates predominant mitral stenosis. As a rule, the mitral opening snap is inconspicuous or absent if regurgitation is the predominant mitral lesion.[7] Occasionally a mitral opening snap is present without demonstrable or audible diastolic murmur. However, obstruction of a grade to warrant valvulotomy is probably rarely present in such cases. There are exceptions. *Recently I suspected mitral stenosis in a man with severe bronchial asthma because of the presence of an opening snap* (Fig. 166). When his asthma improved and presumably his pulmonary vascular resistance was reduced, permitting increased flow across the mitral valve, a typical diastolic rumble of mitral stenosis appeared (Fig. 166).

The mitral opening snap usually persists after mitral valvulotomy. When valvulotomy has succeeded in relieving the obstruction at the valve, the interval between the second sound and the opening snap is increased.

It is doubtful that a normal AV opening sound can be heard or recorded at the surface of the chest. However it may be that such a sound will be demonstrated with regularity by intracardiac phonocardiography (p. 84), especially on the left side of the heart. In a variety of conditions in which stenosis and even fibrosis of the AV valves is absent, a sound suggesting an AV opening snap has at times been heard. Leatham (863) comments on it in atrial septal defect. Luisada (975, p. 276) mentions it in patent ductus arteriosus. Evans and colleagues (444) saw it in a case of primary pulmonary hypertension. I also have found a sound suggesting an opening snap in a case of primary pulmonary hypertension with tricuspid regurgitation and have now seen it in a few cases of high flow across the mitral or tricuspid valve as in patent ductus arteriosus, atrial septal defect, or ventricular septal defect.

[7] In the SLCC a mitral opening snap is often demonstrable in cases of overwhelmingly predominant mitral regurgitation but usually such opening snaps situated between the second sound and the third sound gallop are not evident on auscultation.

TABLE 9

Extra Cardiac Sounds

	Area Heard Best	Subject Heard In	Temporal Relationships	Physiologic Influences	Mechanism	Comments
Split S_1	Apex, LLSB	Bundle branch block, ventricular extrasystoles	Usually 0.05 sec separation		Asynchronism in closure of AV valves because of asynchronism in triggering of ventricles	
Split S_2	Pulmonary, LLSB	ASD, bundle branch block, reduced lung compliance, mitral regurgitation	Up to about 0.08 sec separation	Present only in or increased by inspiration (In left b.b.b. decreased by inspiration)	(1) Discrepancy in stroke volumes of ventricles, (2) asynchronism of ventricular triggering	
S_3 (normal or gallop)	Apex, LLSB	Children, myocardial disease, mitral regurgitation	About 0.14 sec after S_2	Increased by exercise, recumbency, inspiration	Vibration of ventricular myocardium from rapid filling	Distinction of normal S_3 from protodiastolic often difficult in young people
S_4 (atrial sound, normal or gallop)	All areas	Children, left ventricular failure, AV dissociation	0.08–0.14 sec after P wave		Components (1) tensing of atrium (2) movement of blood to ventricle (3) vibrations in wall of ventricle	
Mitral opening snap	All areas, may be least at apex, loudest in left mid precordium	Mitral stenosis	0.06–0.09 sec after S_2	Delayed and less loud after longer diastolic periods in atrial fibrillation	Abrupt billowing of stenotic mitral curtain toward ventricle, sudden check of mitral opening	Often miscalled split S_2
Isodiastolic pericardial snap	Apex, LLSB	Constrictive pericarditis, occasional pericardial effusion with tamponade	0.08–0.12 sec after S_2		Abrupt halt in diastolic filling of the ventricle	Easily mistaken for split S_2
Systolic click, early	Base	Dilated pulmonary artery or aorta, especially with hypertension of corresponding circuit	Up to 0.07 sec after S_1	Loudest in expiration	Snapping of arterial walls early in ventricular ejection	May be confused with split S_1 at base; if S_1 faint at base may simulate snappy S_1
Systolic click, especially mid or late	All areas, least at base	Previous rheumatic fever, chest deformity such as pectus excavatum	Variable	Position of click in systole may vary with respiration	Pleuropericardial adhesions, grating of costochondral or chondrosternal joints	May be multiple late clicks, probably most often due to adhesions; may be mistaken for S_2 and opening snap of mitral stenosis

by aortic stenosis and possibly by *aortic regurgitation*. The interval is shortened immediately *after exercise* because of rise in left atrial pressure and *is prolonged* with administration of norepinephrine because of the elevation of systolic pressure in the left ventricle (64). The Valsalva maneuver is likely to result in prolongation of the S2–OS interval. The S2–OS interval is variable in atrial fibrillation, being longest after longer preceding diastolic periods (Fig. 163).

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In the SLCG the opening snap has the same characteristics *as* systolic clicks (see p. 180): *(1)* short duration, *(2)* tendency to pure frequency content, *(3)* failure of the frequency bottom to reach zero. The differentiation *from the* second part of a *split second* sound is clinched by the demonstration *of* two separate distinct components of the second sound preceding the opening snap. This is, of course, most likely to be possible with inspiration and in recordings from the pulmonary area, where, fortunate for this observation, the opening snap is usually well heard and demonstrated.

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The mitral opening snap usually persists after mitral valvulotomy. When valvulotomy has succeeded in relieving the obstruction at the valve, *the interval between the* second sound and the opening snap is increased.

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CHAPTER 13
Murmurs

NOISY SYSTOLIC MURMURS

Leatham (856) proposes that systolic murmurs be divided into two general categories (*1*) *Ejection systolic murmurs,* which are midsystolic in timing and associated with flow through the aortic or pulmonary valve, (*2*) *regurgitant systolic murmurs* which are holosystolic (p 50) and which are produced by mitral or tricuspid regurgitation or by ventricular septal defect

Ejection systolic murmurs swell to a peak about midsystole and invariably finish before the second heart sound, or at least before that portion of the second sound produced by closure of the valve where the murmur originates This gap is to be expected since forward flow must have ceased before the local backflow responsible for valve closure can occur A murmur of the ejection type originates at the aortic or pulmonary valve under the following circumstances alone or in combination

1 Stenosis of valve or infundibulum
2 Valvular damage without obstruction
3 Dilatation of the vessel beyond the valve
4 Increased flow or rate of ejection

Aortic systolic murmurs are usually best heard in the aortic area However in children with congenital aortic stenosis, the systolic murmur may be loudest at the left sternal border leading to a misdiagnosis of ventricular septal defect The murmur tends to be well heard at the base of the neck on the right and a frequently confusing feature at the apex The presence of a silent interval between the end of the murmur and the second sound is a useful point of differentiation from the holosystolic murmur of mitral regurgitation and ventricular septal defect When the order of closure of the arterial valves is paradoxical as with severe aortic stenosis or left bundle branch block, the murmur may reach the earlier pulmonary component but not the aortic

Aortic stenosis results in a classical murmur which was earlier referred to by Leatham (858) as an *ejection stenosis murmur* In the oscillogram (Figs 167 and 168) the murmur is diamond shaped, that is has its intensity peak in midsystole In the spectrogram the murmur has the configuration of a Christmas tree since its frequency peak is likewise in midsystole

Aravanis and Luisada (26 27) find that the peak of the murmur—they used intensity peak but the same statement should apply to frequency peak—is later in systole the severer the grade of aortic obstruction In fact many of the murmurs included in the general class of ejection systolic murmurs are virtually decrescendo murmurs, so early does their peak occur after the first heart sound Because of the frequent association of an early systolic click (see p 179) with murmurs of the ejection type the murmur frequently displays a decrescendo fall away from an early systolic click

In aortic regurgitation an ejection systolic murmur, which sometimes is even associated with a systolic thrill results from a ventricular stroke output which is increased in both rate and volume and which is occurring across a deformed albeit not stenotic valve Dilatation of the aorta beyond the valve—in luetic aortitis with aortic regurgitation for example—may contribute It is proper to speak of the murmurs of aortic regurgitation not merely the murmur referring only to the diastolic one

Pulmonary stenosis (Fig 169) is associated with a systolic murmur of a shape similar to that of aortic stenosis but with a peak situated later in systole Because of prolonged right ventricular systole and much delayed pulmonary valve clo

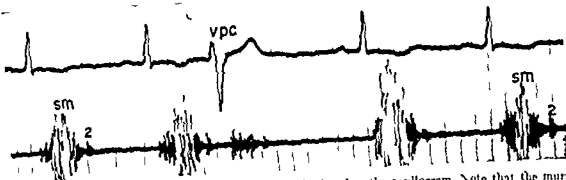


Fig 167 Diamond shaped murmur of aortic stenosis as displayed in the oscillogram Note that the murmur stops before the second sound that the murmur with the extrasystole is much reduced in intensity and that the murmur after the compensatory pause is increased in intensity (Each of the intervals indicated by the time lines is 0 10 sec)

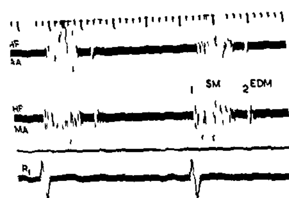


Fig 168 Aortic stenosis
Mid systolic ejection murmur at aortic area (AA) finishing before aortic closure (?) There is an early diastolic murmur (EDM) (Courtesy of Dr Aubrey Leatham)

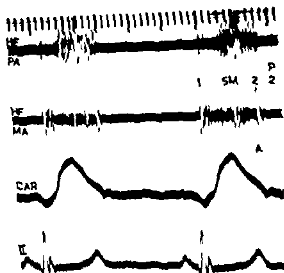


Fig 169 Pulmonary stenosis
RV pressure 65/5 mm Hg Mid systolic ejection murmur goes up to aortic closure sound but ceases before late soft pulmonary closure sound

sure the systolic murmur tends to extend into the aortic closure sound and may extend across and drown it However since it always stops before the pulmonary valve closure the murmur abides by the specification laid down for an ejection murmur

With dilated pulmonary artery an early systolic click is particularly likely to occur and the ejection murmur is likely as stated above to be decrescendo from it With increased flow in the pulmonary artery—as in atrial septal defect and other examples of left to-right shunts before the level of the pulmonary valve thyrotoxicosis exercise complete heart block with increased stroke volume because of the low rate anemia exercise in young subjects—the ejection systolic murmur which has its peak soon after the first sound Here is another demonstration that the timing of the peak of the murmur is largely dependent on how much obstruction there is to ejection

A large proportion of innocent (so called functional) murmurs heard at the base are probably of the nature of ejection systolic murmurs They finish before the second sound become louder with increased blood flow of exertion or excitement and tend to disappear as the child becomes older

The ejection systolic murmur associated with high flow in the pulmonary artery often has a

scratchy quality. In thyrotoxicosis this feature is so striking that the murmur is referred to as a *Lerman Means scratch*. *It may be confused with a pericardial friction rub.* The anatomical superficiality of the pulmonary artery, especially when dilated, is probably in part responsible for the *superficial quality of this murmur.*

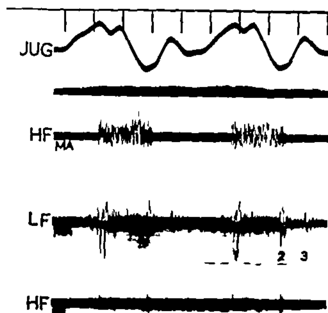


Fig. 170. Mitral regurgitation with holosystolic murmur. There is a protodiastolic gallop in the low frequency *recording (LF). MA = mitral area. HF = high frequency* recording. PA = pulmonary area. (Courtesy of Dr. Aubrey Leatham.)

Regurgitant systolic murmurs are, with few exceptions (see below), holosystolic, because throughout systole pressure in the donor chamber is higher than that in the recipient chamber. Take, for example, the left ventricle and left atrium in mitral regurgitation. There is persistence of a big pressure difference at the time that aortic closure occurs and continuation of the murmur is therefore likely to obscure the second sound—aortic closure sound—at the apex. The exaggerated third heart sound *in mitral regurgitation* must not be mistaken for the second sound.

As to its shape, the murmur of mitral regurgitation (Fig. 170) may be plateau, decrescendo from the first sound, or crescendo to the second sound. However, it is probably for practical purposes always holosystolic. Vaquez (24[illegible]) thought that functional mitral regurgitation could produce a nonholosystolic murmur. One can conceive of an anatomical situation such that the systolic murmur occurs only when pressure in the ventricle is highest and therefore capable of prying the valve open, as it were. This situation is not proved, however, and possibly is unlikely, since

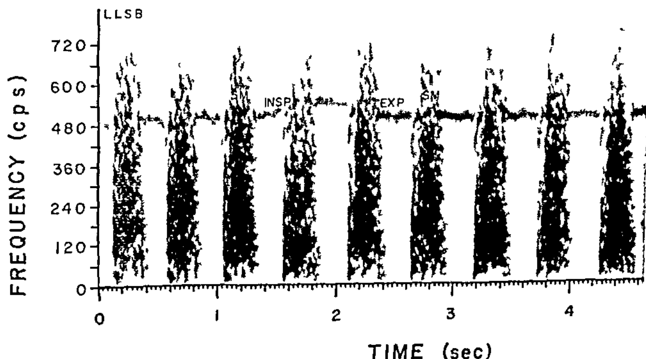


Fig. 171. LLSB in C. A. (757015 B9062) asymptomatic 5 year old male with typical *maladie de Roger* confirmed by cardiac catheterization; pulmonary pressure normal.

ventricular pressure builds up rapidly in the ventricle, is well maintained and falls off rapidly.

The basis for the three different shapes of systolic murmur is unknown. Particularly it is difficult to understand the hemodynamic and anatomical basis for the configuration of the type of regurgitant murmur which has its peak in late systole in the vicinity of the second heart sound. To the ear these murmurs may seem not to be holosystolic, but by graphic means it is possible to demonstrate components of the murmur in early systole. When a murmur is truly limited to the latter part of systole, or if it is circumscribed (with a brief silent gap between it and both the first and the second sound), if it is introduced by a *mid- or late systolic click* and if it varies appreciably with respiration, then differentiation from a regurgitant murmur is provided and extracardiac origin is suspected. The only exception to the statement that circumscribed mid-systolic murmurs with a silent interval both before and after the murmur are not always extracardiac seems to be some cases of aortic stenosis. Especially when left bundle branch block is present, there seems likely to be a gap between the first sound and the murmur as well as the expected gap between the murmur and A.

The holosystolic murmur of mitral regurgitation is loudest at the apex but radiates well into the left axilla and to the back. With mammoth enlargement of the left atrium unusual transmission of the murmur may occur. It may be heard in strikingly loud form in the back and at times as far as the sacrum or even the iliac crests and occiput (1478). Conduction in the osseous structure is probably responsible; the aneurysmal left atrium is likely to impinge on the spine and may rarely erode the vertebral bodies like an aortic aneurysm, sparing the more resilient intervertebral disk. Children and adolescents are most likely to show the phenomenon, indicating that an enormous heart in a small chest is probably the responsible factor. When the large left atrium presents to the right of the sternum with a systolic expansile pulsation and sometimes thrill in that area, there may be heard in the same area a systolic murmur which is almost *certainly* the mitral regurgitant murmur. This creates confusion with other possibilities, especially aortic stenosis and tricuspid regurgitation. The fact that the murmur is loudest at about the level of the third right interspace and is not transmitted into the neck and the demonstration of mammoth left atrium by radiologic techniques help clarify the diagnosis.

Although characteristically the murmur of uncomplicated ventricular septal defect (*maladie de Roger*) is holosystolic (Fig. 171), it becomes limited more and more to early systole when pulmonary resistance is increased and the left-to-right shunt reduced or eliminated. With a *moderate* degree of pulmonary hypertension the abbreviation of the murmur gives it the appearance of an ejection murmur. Another complicating feature is that the dilated pulmonary artery and increased pulmonary flow may produce an early systolic click followed by a genuine ejection murmur.

NOISY DIASTOLIC MURMURS

There are three main varieties of non-musical diastolic murmur: (1) the decrescendo (or diminuendo) relatively high pitched diastolic murmur which begins immediately with the second heart sound; (2) the relatively low pitched rumbling diastolic murmur beginning after the second sound by a short interval—the so-called mid-diastolic murmur; and (3) the presystolic or atriosystolic murmur. These three we will refer to as the *arterial diastolic*, the *AV passage diastolic*, and the *atriosystolic* murmurs, respectively.

Arterial diastolic murmurs result from aortic or pulmonary regurgitation. Because of the relatively high pressure in the aorta normally and in the pulmonary artery in conditions which are associated with the so-called Graham Steell murmur, the arterial diastolic murmur is high pitched, whizzing, whirring in quality.

With rare exceptions the arterial diastolic murmur begins immediately with the closure sound of the valve at which the murmur is generated. Holldack and Wolf (706) observed a silent interval in some cases of pulmonary regurgitation and such an interval may occur in aortic regurgitation (see below). Occasionally it increases in intensity and peak frequency a short time before the decrescendo; that is, it is crescendo-decrescendo rather than merely decrescendo (1529). This pattern doubtless corresponds to an acceleration of re

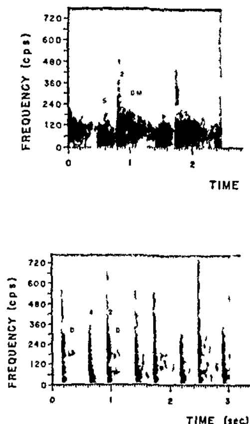


FIG 172 Typical murmur of *aortic regurgitation* decrescendo in peak frequency and in intensity (*Above*) Advanced case The systolic murmur may be caused by aortic regurgitation alone or may indicate a small amount of aortic stenosis (*Below*) Early case

gurgitation momentarily before deceleration It seems that it is regurgitation of mild or moderate degree that is most likely to result in a murmur of crescendo decrescendo pattern The crescendo portion corresponds temporally to the dicrotic wave of the aortic pressure pulse (706) A gap heard on auscultation in some cases of aortic regurgitation may have its basis in the crescendo decrescendo pattern the former phase may not be appreciated (860) In other cases paradoxical splitting of the second sound, with the aortic sound after the pulmonary and possibly reduced in intensity may be the basis In some other cases the basis is by no means clear In these instances there appears to be an entirely silent interval between the second sound and abrupt onset of the murmur about 0 10 or 0 12 sec later Since the mitral valve has opened shortly before with decompression of the left atrium is it possible that support of the aortic ring provided by the contiguous mitral ring is lost so that aortic regurgitation rather abruptly begins or is increased? The decrescendo diastolic murmur of aortic or pulmonary regurgitation is decrescendo not only in intensity, as is well demonstrated by the oscillogram but also in peak frequency as is demonstrated by the spectral phonocardiogram

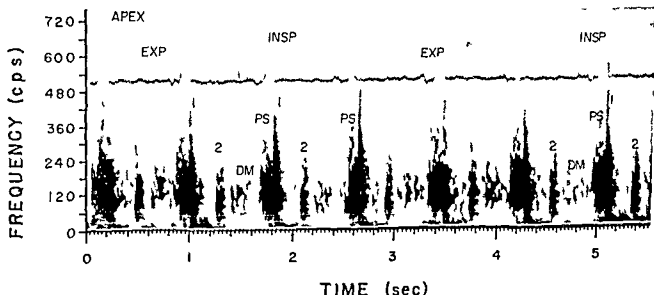


FIG 173 Mitral stenosis

Apex in M K (H L H 94572) 19 year old female There is a minimal systolic murmur An opening snap is not clearly demonstrated here but was evident in LLSB and pulmonary areas M_1 is ringing as indicated by the relatively high frequency content demonstrated and is delayed relative to QRS The two parts of the diastolic murmur—passive first part (DM) and presystolic crescendo (PS)—are clearly demonstrated The presystolic murmur has the appearance of half the Christmas tree which is characteristic of an ejection stenosis murmur as in aortic stenosis

The arterial diastolic murmur of aortic origin (Fig 172) is most often loudest at Erb's point (the third left intercostal space at the sternal border) occasionally at the cardiac apex and sometimes in the left axilla (the Cole Cecil murmur p 27) Balthazar Foster (p 29) thought that the direction of radiation of the aortic diastolic murmur was an indication of the precise anatomical change at the aortic valve particularly of which cusp was predominantly involved More recent analysis of this matter by Kerr and Palmer (782) (p 279) make it seem unlikely that much diagnostic significance should be attached to the direction of radiation of the murmur, although Harvey (632) was impressed with the predominant radiation down the right sternal border in cases of syphilitic aortitis the Marfan syndrome and coronary sinus aneurysm Hoffman and Blount (690) and White (1548) also wrote that in syphilis radiation was down the right sternal border more commonly than in rheumatism

The arterial diastolic murmur of *pulmonary* origin is usually confined to the left sternal margin From the standpoint of quality there is really nothing to differentiate it from the murmur of aortic regurgitation Any arterial *diastolic* murmur heard at the right of the sternum is probably of aortic origin Furthermore the murmur of pulmonary regurgitation may on rare occasion be influenced by respiration that is it may be accentuated in late inspiration As noted above Hollduck and Wolf (706) *observed a gap between S_2 and the murmur in some cases* Usually however ancillary signs such as the peripheral signs of aortic *regurgitation* must be relied upon to make the differentiation

The most frequent form of pulmonary regurgitation is that which generates the Graham Steell murmur the murmur of high *pressure in the* pulmonary artery (p 30) *Only very rarely* does disease of the pulmonary valve producing pulmonary regurgitation *occur in the absence of* pulmonary hypertension When such does occur for example with an isolated congenital *defect of* the pulmonary valve or damage by *gonococcal* endocarditis the murmur is likely *to be more low* pitched and blubbering

Both types of arterial diastolic murmurs are best heard when the subject is in the sitting position especially if he is *leaning forward in full* expiration The examiner may even resort to placing the patient in the knee chest position *These murmurs* are notorious for being faint and easily missed

The passive AV *diastolic murmur accompanies* stenosis of the mitral (Fig 173) or *tricuspid* valve relative or absolute It is always separated from the second sound by an interval It may, in the case of organic stenosis begin with an opening snap or even in that instance may have its onset somewhat later than the opening snap The passive diastolic murmur is usually decrescendo in pattern If the mitral stenosis is sufficiently *mild*, diastole sufficiently long, *or flow* sufficiently low, the murmur may actually end before the onset of the atrial systolic murmur or before the end of diastole in the case of atrial fibrillation The *decrescendo* character and *the recession of the murmur* are the result of diminishing atrioventricular pressure *differential The* simultaneously recorded pressure curves from left atrium and left ventricle obtained by left heart catheterization, sometimes indicate that the reduction of the gradient is more a matter of rise in ventricular pressure than of fall in atrial pressure (659) This would be expected to be the case particularly *if* there were associated aortic regurgitation *or* change *in the pressure* volume characteristics of *the left ventricle* resulting from the ventricular hypertrophy of an associated lesion such as aortic stenosis Elevated left atrial pressure may be maintained by persisting inflow from the lungs whereas ventricular pressure will *rise unusually* rapidly for the reasons outlined

When the stenosis is of the relative type—that is the valve is normal but flow *across* the orifice is increased and the ventricle beyond it enlarged—then the passive AV diastolic murmur *tends to be introduced* by an exaggerated third heart sound The particular variety will be referred to as the *Carey Coombs* murmur because although the eponym was originally assigned to the type of murmur occurring specifically *in the course* of acute rheumatic fever the murmur of all relative mitral stenosis has the same pattern In general the passive AV diastolic murmur is low pitched and *rumbling* in quality The Carey Coombs *murmur* often may be best described as blubbering Flow of blood into the ventricle in

TABLE 10

A Comparison of the Arterial Diastolic Murmur of Aortic Regurgitation with the Passive Diastolic Murmur of Mitral Stenosis

	Arterial Diastolic Murmur of Aortic Regurgitation	Passive Diastolic Murmur of Mitral Stenosis
Time	Early, immediately following the second sound	Later, beginning an appreciable interval after the second sound, often immediately after the opening snap when it is present; *it extends through* mid-diastole if loud; if the stenosis is *well marked* and the rhythm normal, it is followed by a presystolic accentuation and thrill, especially when the pulse is quickened after exercise
Character	Blowing, often high pitched	Rumbling, usually low pitched, rarely blowing
Site	Maximal along the left border of the sternum, heard frequently at the apex and rarely maximal at the aortic area (in such a case luetic aortic dilation is usually present)	Maximal at the apex and often limited to a very small area, but sometimes heard as far as the sternum
Position of body	Best heard in the *upright position* leaning forward	Best heard in the *recumbent position*
Stethoscope	*Best heard with the* diaphragm; sometimes it may be *heard better with* the naked ear	*Best heard with the* bell chest piece

the first part of diastole seems to be especially likely to produce sound in children. Furthermore, a murmur is especially *likely to be generated* at the *mitral* valve in hypervolemic states. This may be the result of higher pressure in the left atrium, which in turn is due to its steeper pressure-volume curve than in the right atrium. However, high flow does generate a passive diastolic murmur at the tricuspid valve in atrial septal defect.

What will be said about *the locus of maximum* audibility of the presystolic atriosystolic murmur (see below) applies equally to the passive AV diastolic murmur.

By way of summary, reference can be made to Table 10, which was originally devised by Paul Dudley White in 1926 (1510).

The *atriosystolic murmur* is probably better so termed, rather than presystolic, since when there is some degree of atrioventricular dissociation the murmur may not be immediately presystolic (1150). The atriosystolic murmur is essentially an *ejection systolic murmur (see p. 194)*. When the PR is prolonged so that the atriosystolic murmur stands alone, it is seen that it has the diamond shape in the oscillogram and in the spectrogram has the configuration of the Christmas tree, just as *does the much louder murmur of aortic stenosis*. When the PR interval is of normal duration the presystolic murmur is half the Christmas tree of an ejection systolic murmur, being cut short by the snapping mitral first sound.

Excluding the Carey Coombs murmur (p. 199), which is a different matter, the presystolic murmur is often the sole murmur of minimal mitral stenosis. The reason is obvious: it is to be expected that, in mild cases, only with atrial systole will there be sufficiently rapid transmitral *flow to produce* a murmur.

In the past some (e.g., 901) have questioned whether the presystolic murmur is truly crescendo. One group (13) states as follows: "The acoustic crescendo effect is usually an *auditory* illusion because of its close proximity to the accentuated first sound." They have been led to this conclusion by failure to demonstrate a clear crescendo of intensity in the oscillographic PCG (p. 296). *The spectral PCG leaves no doubt that* the presystolic murmur of mitral stenosis is indeed *crescendo*.

The atriosystolic origin of the presystolic murmur, a matter of debate half a century ago, has again *been brought in question by one group* (1143) which claims that it is in fact a murmur

produced by vibrations of the mitral valve early in ventricular systole as the mitral valve moves toward the atrium. Others have conceived of there being regurgitation at the mitral valve for a brief period before the diaphragm of the *stenotic valve* snaps toward the atrium with production of the ringing first heart sound. Both theories are directed at the idea that the murmur is systolic, not presystolic. The fallacy of this view, which has no definite evidence to support it in either of its two *forms*, is revealed by recording of the murmurs in mitral stenosis which show the presystolic murmur beginning well before the QRS of the electrocardiogram. In 1913 Sir Thomas Lewis (904) wrote: "... it is generally believed to be presystolic in time, yet from time to time its actual position in the cycle has been hotly contested. I do not propose to pursue a discussion which at the advent of sound records has become almost purely historical." The isolated presystolic murmur with long PR interval is further evidence for the atriosystolic nature of the presystolic.

In mitral stenosis the two AV diastolic murmurs just discussed—passive diastolic and atriosystolic—are audible at the cardiac apex, often in a confined area, however. They are best audible when the subject is *in the left lateral decubitus* position, possibly because *the apex is* brought into more intimate contact with *the anterior* chest wall.[1] The exercise of sitting up *and lying down* several times and then turning on the *left side* will often bring out these murmurs when *they* are not otherwise audible. Walking about will *sometimes* bring out the murmur of mitral stenosis when sitting up does not. In a very ill patient merely having him cough may be sufficient to bring out a *mitral diastolic* murmur. Pharmacologic *methods*, e.g., amyl nitrite and phenylephrine, have sometime been used to increase flow to the point that the murmur will be *heard*. The presystolic murmur is likely to be increased by any increase in heart rate—probably because the atrium had less time for decompression before atrial contraction occurs—and the effects of many measures may be mainly through the effect on heart rate. There may in effect be a summation of the passive *diastolic* and the atriosystolic murmurs: blood flow across the mitral valve may actually fall.

As a rule the diastolic murmurs of mitral stenosis do not radiate widely and are audible only in a very limited area. They usually do not radiate into the left axilla as in the case of the systolic murmur of mitral regurgitation. Occasionally, however, when very loud, they may be audible over most of the *body of the heart and even in* the aortic area. In children *and other individuals* with small chests this is especially likely to be the case.

The location of the *diastolic murmurs of tricuspid* stenosis is more variable. They *may be best* heard at the lower left sternal *border, in the region* of what is considered the cardiac apex, or at any *point in between*. Differentiation from the diastolic murmurs of mitral stenosis is often difficult. *Accentuation* by inspiration is helpful. Occasionally, however, the diastolic murmurs of mitral stenosis are accentuated by inspiration. The diastolic murmurs of organic tricuspid stenosis—disregarding for the moment the functional tricuspid stenosis of atrial septal defect—tend to be louder than the corresponding murmurs *of mitral* stenosis, are often accompanied by a very striking thrill at the left of the sternum and in the epigastrium, have a greater frequency span than usually seen in the murmur of mitral stenosis. This last does not mean, however, that the *murmur* sound higher pitched. These *features* of the murmurs of tricuspid stenosis are probably the result of the more superficial location *of the tricuspid* generator.

Rytand (1334) described what he termed an auricular *diastolic murmur with complete* heart block in *elderly patients*. It is preceded by an atrial heart *sound*.

An *interesting early* diastolic murmur at the apex *may occur* in patients with 2:1 heart block and a slow ventricular rate. The murmur has the characteristic of a Carey Coombs murmur. The first unconducted P wave occurs early in the ventricular diastole. The combination of atrial systole and rapid passive inflow is probably responsible for the murmur. The second P wave

[1] *The precise mechanism is unknown. One suggested possibility is that in the left lateral decubitus gravity favors flow through the mitral valve. Harvey (692) has even questioned that there is anything special about the left lateral decubitus and suggests that the exertion of turning into that position may alone account for the temporary accentuation of the diastolic murmur.*

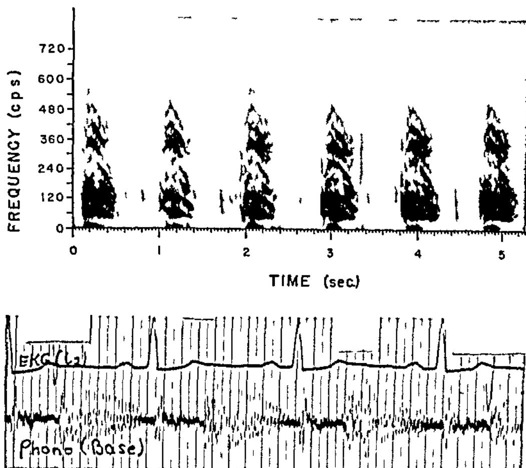


FIG 174 Recordings of a "sea gull" diastolic murmur of *retroverted aortic cusp* (*Above*) Spectrogram (*below*) Oscillogram made with a Hathaway galvanometer with a natural frequency of about 1000 c p s Note that in the oscillogram it is possible to identify the second harmonic at a frequency twice that of the more intense fundamental

which may be followed by a ventricular contraction after a normal PR interval, may have no accompanying murmur

MUSICAL MURMURS[2]

Murmurs are musical when to the ear they present a quality which is perhaps best defined by the improved ability to represent the murmur by conventional musical notation It is easier to place a musical murmur on a musical scale In the oscillogram, they display regular vibrations at the frequency of the fundamental although occasionally (see Fig 174B also see 1529, Fig 32) the second harmonic is also discernible In the spectrogram musical murmurs are characterized by the presence of harmonics, a feature which constitutes the most valid and objective definition of musicality (Fig 174)

Systolic

Musical systolic murmurs heard over the precordium are of three main types (1) the variety heard in calcific aortic stenosis (Fig 175), (2) that of mitral regurgitation and (3) the musical pleuropericardial sound Each of the three has sufficiently distinctive features that confusion is unlikely

In *calcific aortic stenosis* one is likely to hear in the aortic area itself and at the base of the neck on the right a noisy non musical murmur with characteristic diamond or Christmas tree configuration by phonocardiogram At the left sternal border left midprecordium and apex the murmur may be strikingly musical (161, 521) In the SPCG from these sites there are demonstrated conspicuous chevron shaped harmonics which have their apices at the point in systole where the peak of the noisy murmur is located It seems likely that the noisy murmur is produced by the jet in the aorta, whereas the musical murmur is related to the regular periodic and therefore musical vibrations of the stenotic valve diaphragm (Fig 176) It is useful to refer to this dissociation

[2] See reference 1083

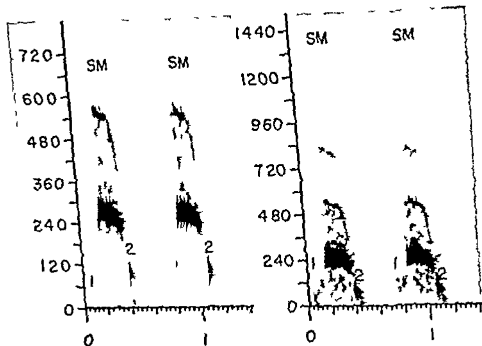


FIG 1 5 (*Above*) Systolic murmur of *calcific aortic stenosis* (pulmonary area) Both musical and noisy elements are present giving a rasping quality to the murmur The murmur has the Christmas tree shape characteristic of aortic stenosis (*Below*) Systolic murmur in *calcific aortic valve disease* The murmur was loud and quite musical everywhere and there were no peripheral signs of aortic obstruction Probably in this case there was fibrosis and calcification of the individual cusps with minimal adhesion at the commissures and very little obstruction to systolic ejection

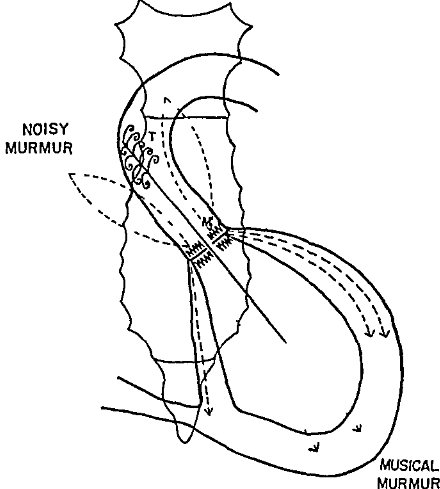


Fig. 176 Dissociation of noisiness and musicality in murmur of aortic stenosis—the Gallavardin phenomenon. In aortic stenosis, especially calcific aortic stenosis, the murmur may be musical at the apex and left midprecordium, noisy at the aortic area and base of the neck on the right. The musical component may arise in the stenotic valve and the noisy component higher in the aorta in connection with the jet. The jet noise in other situations is highly directional and zones of maximum intensity such as those indicated by the "rabbit ears" in dashed lines are theoretically to be expected.

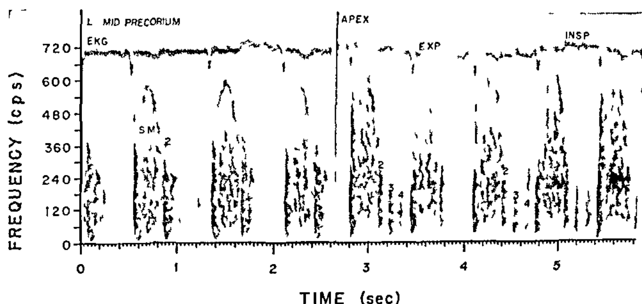


Fig. 177 Musical systolic murmur of mitral regurgitation.

Left midprecordium and apex in L. L. (610441), 18 year old female who had several attacks of acute rheumatic fever, the most recent six years before. She is essentially asymptomatic. The musicality of her systolic murmur is most striking toward the *left sternal border. There is a holosystolic murmur which especially in (A) has a harmonic* at about 500 c p s. At the apex (*B*) there is a protodiastolic gallop typical of mitral regurgitation and either a presystolic gallop or short murmur.

of quality in aortic stenosis is the Gallavardin phenomenon.

Frequently the harsh musical systolic murmur best heard at the apex is misinterpreted as being generated at a calcified mitral valve (536 Fig 3 B). Although such a valve is occasionally the cause of a musical systolic murmur, the oscillographic and spectrographic patterns are distinct. Specifically, the murmur is likely to extend throughout systole as in most murmurs of mitral regurgitation. The harmonic pattern is different as a less specific distinction.

At the apex a delicately musical murmur of *mitral regurgitation* (Fig 177) is frequently heard especially (1) during the active stage of acute rheumatic carditis (2) as a more permanent sequel of bacterial endocarditis (3) with a heavily calcified mitral valve, and (4) in patients with the Marfan syndrome, possibly because of redundant chordae tendineae. The frequency level of the harmonics in this murmur are usually higher than those in other types of musical murmurs. Often, there is only a single harmonic, i.e. the fundamental present.

The systolic murmur of ruptured papillary muscle or chorda tendinea is described as loud, rough, harsh and coarse (44). Occasionally (1369) the adjective *musical* is applied to it; that such is not more often done is probably the result of differing ideas of what constitutes musicality.

As a general rule the more obtrusive a symptom is the more it impresses the mind of the observer and much more importance is attached to it than to less conspicuous phenomena. This is particularly noticeable in

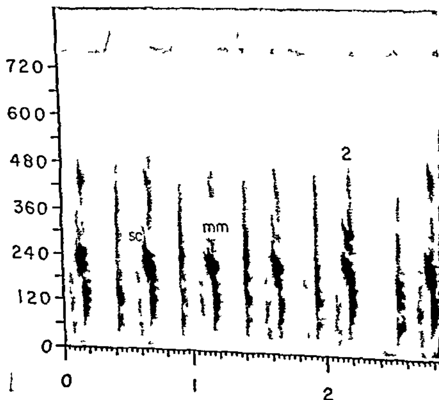


Fig 178 Musical extracardiac murmur

This musical late systolic murmur developed 18 months before this recording and following a respiratory infection in J W (73406) a 6 year-old girl. The murmur was preceded by a systolic click, tended to extend over the second sound slightly, was maximal in inspiration, was exaggerated with excitement and exertion to the point of being audible at a distance from the body. It was compared by the parents to the sound of a rusty hinge. It is probably a violin type of pleuropericardial rub. This patient had mild pectus excavatum which may have contributed to the genesis of the murmur.

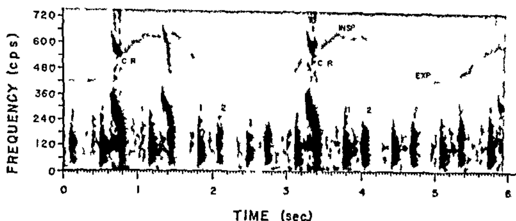


Fig 179 Musical extracardiac murmur late in systole (C R) occurring only in inspiration in patient V W (106004) with cardiomegaly She had systemic arterial hypertension and multiple pulmonary emboli and probably had had rheumatic fever This is probably a musical pericardial or pleuropericardial rub of the violin family of musical murmurs This sound was present for the three years that the patient lived after the recording shown here

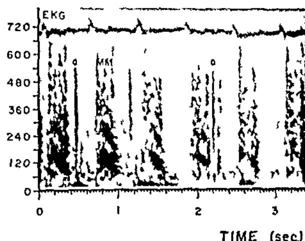


Fig 180 Musical systolic murmur present at the apex and mainly in inspiration in C J (721562) 43 years old during a bout of congestive heart failure of obscure basis

signs which are detected by auscultation To the human mind sounds arising from obscure causes have always been a source of mystery and the human imagination when dealing with the mysterious invariably associates *it with something malign*

Sir James Mackenzie 1908

Although one might cite several varieties of murmur to which Mackenzie's comment is applicable it seems especially apropos to the type of musical systolic murmur next to be discussed—the musical pleuropericardial murmur

A *striking musical* murmur suggesting a whoop (as in whooping cough) or a squeak is an occasional *sequel of pericarditis* (Figs 178 to 180) Its mechanism seems to be analogous to that of the violin group of instruments *This sound persists* for years It is probably more likely to occur when there is pectus excavatum (Fig 178) and/or when the heart is enlarged *It is exaggerated by* anything which increases the vigor of the heart beat *Like most musical murmurs* which as a class include some of the loudest sounds generated in the cardiovascular system this murmur may be audible at a distance One mother heard it sounding like a rusty hinge in her six year old daughter after the child had been disciplined The sound is in late systole as a rule, is often introduced by a systolic click or at times may be replaced by a systolic click, and tends to vary with respiration, being probably most often loudest in inspiration Either full inspiration or full expiration may *eliminate it In phonocardiograms it can at time* be shown that the murmur extends across the *second heart sound, a feature arguing for its* extracardiac origin The main significance of this sound is its lack of significance It is easy *to* ascribe grave significance to it if the phenomenon described is not familiar A sound audible to the unaided ear at a distance from the subject is likely to have frightening connotations for the patient his family and indeed his physician

Looking back over the medical literature one can find reports of musical murmurs which were almost certainly of extracardiac origin One of the earliest is that of Maclachlan (cited by Magdelaine) who in 1843 described a murmur like the bark of a young dog the valves were found to be normal at autopsy In 1880 Osler (1168) reported on an apparently normal 12 year old girl who at

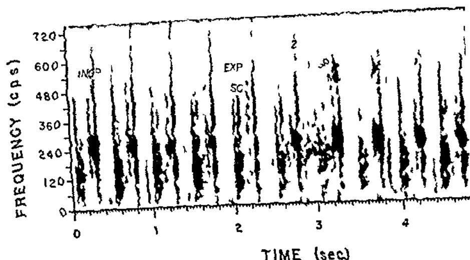


FIG. 181. Musical extracardiac murmur

Apex in C. S. (B2805) 6 year-old female ten days after onset of acute grippal illness. Note the variability of the late systolic murmur which was most intense in inspiration. There is an indistinct systolic click introducing the musical murmur. An early systolic click is loudest in expiration. Eight weeks later the musical murmur was heard only faintly and after vigorous exercise whereas both an early and a late systolic click were now striking.

certain unpredictable times emitted a loud systolic murmur which was distinctly audible at a distance of three feet two inches by measurement and could be heard at any point on the chest and on top of the head. In his *Traité des Maladies du Coeur* Barié mentioned a 71 year old man whom he had seen in 1874 and who had a rasping systolic murmur audible four meters from the chest. Having been awakened from sleep by it when it first appeared the patient thought at first there were large flies in the room. The heart valves were later found to be normal.

One of the most detailed analyses of the musical extracardiac murmur is that of Luan and colleagues (944). They noted two types: (1) the groaning functional midsystolic murmur loudest at the left sternal border[1] and (2) the late systolic murmur introduced by a click, often audible at a distance, extending into early diastole, whining in quality, maximal in the sitting position, often accompanied by a thrilling (*chatouillement*) which the patient can himself feel. They noted that the latter type could be associated with heart disease and cardiac enlargement and granted that the extracardiac origin can be stated only with some reservations in such cases. They thought that this type of murmur is usually maximal in expiration and that it is produced by pressure on a lappet of lung with movement of air through narrowed bronchioles. However, a later communication suggested that he had revised the latter view (917) and considered the murmur pleuropericardial in nature.

In a 33 year old female patient with primary pulmonary hypertension Levine and Harvey (888, p. 262) described a loud leathery grade VI late systolic murmur which was produced during the Valsalva experiment. The patient heard it herself and found that she could reproduce it at will by holding a deep breath, especially if she voluntarily compressed her chest at the same time. It promptly disappeared when the breath was released. Only the usual findings of primary pulmonary hypertension were described at autopsy.

Warburg (1008) described (with phonocardiogram) a murmur similar to that in Figure 180 in a man with mitral stenosis, atrial fibrillation and frequent bouts of congestive heart failure. With an obscure febrile episode the patient noted a noise in the chest which was also evident to the patient's wife. I was able to verify his statement. At every heart beat a clicking or slightly sonorous sound was audible in the room. A phonocardiogram showed that besides the sounds

[1] As will be discussed later (p. 244) this murmur may not be of extracardiac origin.

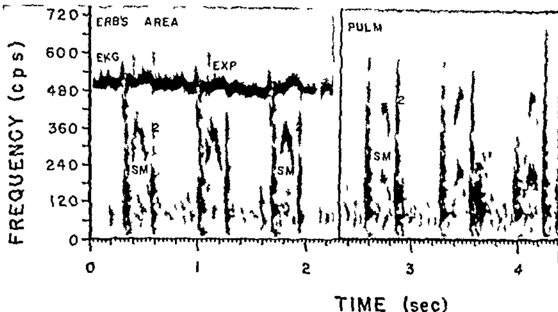


Fig 182 Fig 183

Musical extracardiac murmur?

L L H (776301) 12 year old boy displayed a musical systolic murmur only after exercise and mainly with a mid respiratory position of the chest 182, Erb's area after exercise 183, pulmonic area after exercise with full expiration EKG omitted for better definition of upper harmonics Under different circumstances and even from cycle to cycle with what seems the same circumstances there are striking changes in the frequency level of the harmonics All examinations including cardiac catheterization yielded normal finding (Trigonoidation at the pulmonary valve i e, that this is an exaggerated still murmur is an alternative possibility)

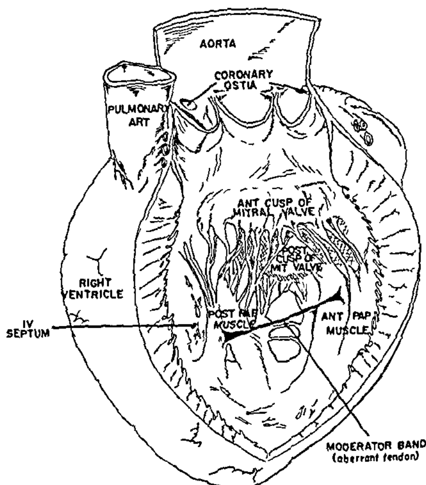


Fig 184 Sketch showing location of *aberrant tendon* (moderator band) in the left ventricle (After Huchard (718) Rolleston (1308) described a similar specimen

ually found in the patient there was a new murmur partly during the systole partly immediately after the systole It was quite clear that the new sound included higher frequencies than the other sound

Levine and Harvey (888 Fig 2[illegible]) described a 42 year-old woman with rheumatic valvular disease in whom a very peculiar weird sounding rough musical murmur was occasionally present in addition to her usual murmurs of mitral stenosis and regurgitation and aortic regurgitation The murmur was usually absent at rest, was always present immediately after brief exercise and disappeared again with rest It could be heard a foot away from the chest It is known to have been elicitable for at least four years and was late systolic in timing running over S[illegible] Atrial fibrillation was present

Weber ([illegible] p 29) described and illustrated a musical late systolic murmur which occurred in a locomotive engineer who sustained a severe chest contusion in a railroad accident The murmur was

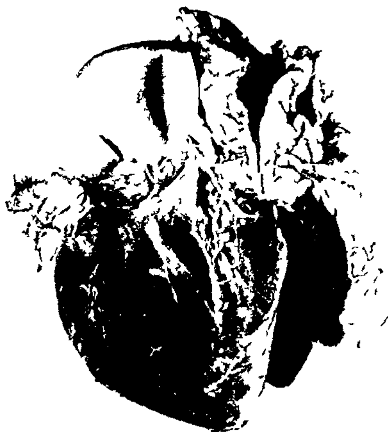


Fig 18[illegible] Aberrant tendons of ventricle

The heart in M V (1[illegible]) colored female who died at the age of 43 years having been seen in this hospital twenty years previously at which time her heart sounds were normal Four years before death she developed toxemia of pregnancy Thereafter hypertension was persistent and heart failure recurrent During the period of toxemia and [illegible] during episodes of heart failure she was found to have a loud harsh musical systolic murmur accompanied by thrill maximal in the left midprecordial area She had noted a buzzing sensation The murmur was widely audible even over the head and sacrum At autopsy the heart weighed [illegible]60 gm All chambers especially the left ventricle were greatly dilated The aortic valve was normal and no structure was found to account for the musical murmur except a net of aberrant tendons traversing the lower part of the cavity of the left ventricle The net consisted of a central portion about 0.5 cm long in its fixed state at each end of which were attached two tendons each about 1.2 cm in length

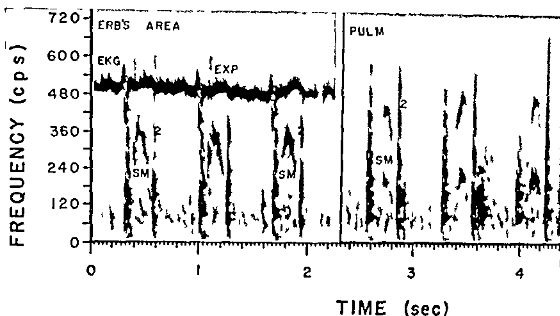


Fig 182 Fig 183

Musical extracardiac murmur?

I L H (776304) 12 year old boy displayed a musical systolic murmur only after exercise and mainly with a mid respiratory position of the chest 182 Erb's area after exercise 183 pulmonic area after exercise with full expiration EKG omitted for better definition of upper harmonics Under different circumstances and even from cycle to cycle with what seems the same circumstances there are striking changes in the frequency level of the harmonics All examinations including cardiac catheterization yielded normal findings (Trigonoidation at the pulmonary valve i e that this is an exaggerated still murmur is an alternative possibility)

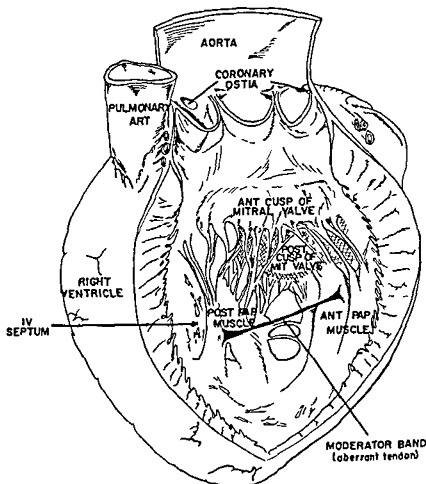


Fig 184 Sketch showing location of *aberrant tendon* (moderator band) in the left ventricle (After Huchard (718) Rolleston (1308) described a similar specimen



FIG 185 Examples of Chiari nets of the right atrium from Chiari's publication (259)

located in late systole and was often introduced by a systolic click. At times only the click remained.

Rare examples of musical murmurs or of musical contribution to noisy murmurs are produced by *aberrant tendons*, such as those sometimes called "moderator bands,"* which may cross the stream of flow of blood and be actuated to musical vibration in a manner comparable to an Aeolian harp. Huchard (715) described the clinical picture associated with aberrant tendons of the ventricle (Fig 184). The main feature is that the musical murmur is usually not present from birth despite the congenital nature of the aberrant tendon, but appears when the ventricle becomes dilated because of some unrelated strain such as systemic hypertension. Only when the aberrant tendon is pulled taut by ventricular dilatation is it possible for a musical tone to be produced. For example, Huchard's first case was that of a 49 year old man seen in 1892. Cremial signs of severe arterial hypertension, cardiomegaly and heart failure were present. The conventional murmur of mitral regurgitation was heard at the apex and in the left axilla. More medially located, maximal in the area of the xiphoid but widely heard, especially to the right of the sternum and up the course of the aorta, was a purring or snoring systolic murmur ("une sorte de piaulement, de ronflement, de bruit de guimbarde"). Unfortunate to the differential diagnosis is the fact that extracardiac musical murmurs may occur in the same clinical setting and display the same characteristics, although usually more variation with respiration will be demonstrated.

Aberrant tendon of the ventricle is rare. The case presented in Figure 185 is that of a patient seen in this hospital twenty years ago with pathological studies by Dr Arnold R Rich. It is probable that the Huchard murmur was present in this case.

There is one more common situation in which aberrant tendon may *contribute* a musical quality to a murmur otherwise noisy. In ventricular septal defects anomalous tricuspid chordae tendineae may insert at the upper and lower margins of the defect (Fig 186). These are probably responsible for the harmonics which are occasionally demonstrated by SPCC in the Roger murmur. Probably especially in the AV communis malformation (Fig 187) is the ventricular defect likely to be so related to tricuspid valvular structures that a musical tone is produced.

Closely akin to the musical murmur of moderator band and aberrant chordae tendineae is that

* The term was assigned in 1837 by King (796) who thought the structure functioned in moderating or checkreining dilatation of the ventricle



Fig 186 Aberrant tricuspid chordae tendineae stretching across right ventricular aspect of high interventricular septal defect and probably responsible for the musical modality of the resulting murmur Such cords are often the site of SBI

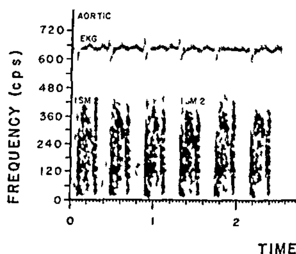


Fig 187 Sounds typical of most areas of the precordium in D M T (B6145) 3 year old white female with what is thought to be A V *communis* There is a striking musical element in the systolic murmur probably caused by anomalous chordae

loud and widely audible Atrial fibrillation was present

Geckeler *et al* (532) presented spectrograms of a musical systolic murmur referred to by them as a grunt, which was heard in the second right intercostal space for about ten days after mitral commissurotomy The authors did not think it to be pericardial in origin however this would appear to me to be the best possibility another being that some vibrant structure such as part of the anterior (aortic) leaflet of the mitral valve or a chorda tendinea was torn loose and was thrown into vibration with ventricular ejection The disappearance of the murmur after ten days would be difficult to explain in the latter case

Laubry and Lebranc (845) described a musical midsystolic murmur introduced by a click in a patient recovering from lung abscess

Harvey (652) has described the clinical and phonocardiographic features of what he chooses to term a cardiac whoop The subject may be perfectly well (one of his cases was a member of the hospital's resident staff) and the sound may be present for an indefinite period The intensity of the murmur tended to wax and wane with respiration The vibrations which constituted it were periodic i e musical The murmur was

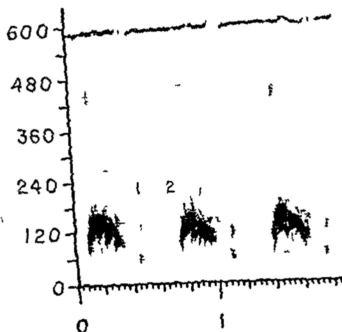


FIG. 190. Musical diastolic murmur of retroverted aortic cusp. In this instance, rather than being accentuated with atrial systole (as in Figure 89) the murmur stops abruptly with atrial systole.

of these harmonics in the same general range of frequency as the natural frequency of the thorax. As will be discussed later (see p. 477) the natural frequency of the male thorax is usually of the order of 120 c.p.s. The fundamental of these murmurs is often in this range, and that which is conducted best to the cardiac apex. The result is a murmur different in quality through the absence or attenuation of many of the overtones.

The musical aortic diastolic murmur produced by rheumatic fever or bacterial endocarditis seldom displays such pure musicality or such well developed harmonic pattern. Most often it is a matter of one or two harmonics in the midst of an otherwise conventional noisy decrescendo murmur. Geckeler and his colleague (532) have presented an exception to this generalization, i.e., a patient with presumed rheumatic heart disease and a full blown musical aortic diastolic murmur. Furthermore we have had what is probably an identical experience.

In patient P. C. (424089) a very loud cooing diastolic murmur with thrill, typical of retroverted cusp, was heard in 1947 when the patient was admitted for left sided heart failure. Although there was no history of syphilitic infection or treatment for the same and the serologic tests (including treponemal immobilization test) were always negative, syphilis was considered likely. During the following year the aortic diastolic murmur became a conventional one with the usual quality, with no very unusual intensity and no thrill. The patient has done quite well on the whole, a consideration which with the others mentioned makes rheumatism the probable basis of the sea gull murmur in this patient. The benign course makes syphilis very unlikely.

In some patients (e.g. T. S. 103183) musicality may appear in a murmur previously conventionally noisy. In others (cf. Figs. 189 and 192) the musical element may disappear leaving an aortic diastolic murmur of the usual noisy quality. The variations in the graphic character of musical aortic diastolic murmurs are many and include differences in the following characteristics:

1. The time interval between the beginning of the murmur and the frequency peak.

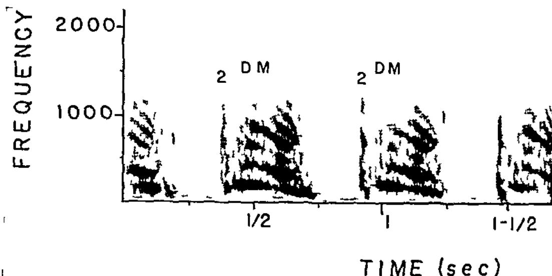


Fig. 189 Retroverted aortic cusp

Typical musical aortic diastolic murmur in patient with syphilitic aortitis. Note intensification of murmur without rise in frequency level of its harmonics toward end of diastole. Probably atrial systole by displacing the retroverted element into the regurgitant stream increases its excursion of vibration without changing its frequency of vibration. When this patient was restudied three years later only a diastolic murmur of conventional noisy quality was found.

which accompanies Chiari's network (Fig. 188) of the right atrium (see p. 410). This is by no means a rare finding at autopsy—2 to 3 per cent according to Yater (1598), 1.5 per cent according to Helwig (669). However, it is much less common for a musical murmur to occur in association. In two cases a continuous musical murmur has been described (18) (1571). It is probable that as with ventricular bands not only does the Chiari network need to be properly oriented in relation to the venae cavae, but also dilatation of the atrium with tensing of the network favors development of such a murmur.

Diastolic

A *musical diastolic murmur* is rarely heard except in one circumstance—retroversion or some similar deformity of an aortic cusp (82, 537, 606, 1378). The usual cause of retroverted aortic cusp is syphilis, but it has in recent times been recognized that cystic medial necrosis of the aorta of idiopathic type may be accompanied by this accident. Occasionally the aortic diastolic murmur of aortic regurgitation in the *Marfan syndrome* is musical although more often it is a conventional noisy murmur. A musical aortic dias*tolic murmur* may be heard with bacterial endocarditis and occasionally with the valvulitis of acute rheumatic fever and with chronic rheumatic valvulitis.

Most features previously described for noisy aortic diastolic murmurs are found in musical aortic diastolic murmurs (Figs. 189 to 191). In addition the murmur of retroverted aortic cusp is as a rule intense. It has the quality of wood sawing or possibly of the cry of a sea gull or other bird.[5] It has been compared also to the sound made by a young frog (1392). The ingenious metaphors are legion. More characteristically than in the case of noisy aortic diastolic murmurs the musical counterpart is *crescendo decrescendo*. The harmonic shows a rise in frequency before the beginning of the decline. This pattern is in large part responsible for the oo of the cooing murmur. It is related to an acceleration then a deceleration of the regurgitant stream with corresponding change in the rate at which the retroverted cusp is driven.

There tends to be a change in quality of the murmur of retroverted aortic cusp on transmission to the apex. The basis is a preferential conduction

[5] Actually experts on bird calls say these sounds resemble those of no birds known to them! Therefore the designations "sea gull" murmur and "cooing dove" murmur for members of the musical group may not be appropriate.

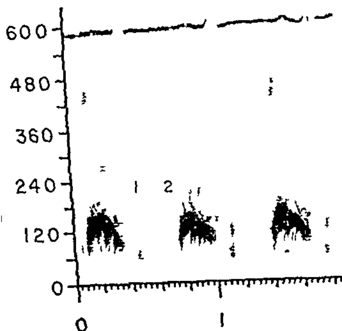


FIG. 190 Musical diastolic murmur of *retroverted aortic cusp*. In this instance, rather than being accentuated with atrial systole (as in Figure 189), the murmur *stops abruptly with atrial systole*.

of those harmonics in the same general range of frequency as the natural frequency of the thorax. As will be discussed later (see p. 477), the natural frequency of the male thorax is usually *of the* order of 120 c.p.s. The fundamental of these murmurs is often *in this* range and is what is conducted best *to the* cardiac apex. The result is a murmur different in quality *through the* absence or attenuation of many of the overtones.

The musical aortic diastolic murmur produced by rheumatic fever or bacterial endocarditis seldom displays such pure musicality or such well-developed harmonic pattern. Most often it is a matter of one or two harmonics in the midst of an *otherwise* conventional noisy decrescendo murmur. Ceckeler and his colleagues (332) have presented an exception to this generalization, i.e., a patient *with* presumed rheumatic heart disease and a full-blown musical aortic diastolic murmur. Furthermore, we have had what is probably an identical experience.

In patient P. C. (424080) a very loud cooing diastolic murmur with thrill typical of retroverted cusp was heard in 1947 when the patient was *admitted for left-sided* heart failure. Although *there* was no history of syphilitic *infection* or treatment for the same and the *serologic* tests (including *treponemal* immobilization test) were always *negative*, syphilis was considered likely. *During* the following year the aortic diastolic murmur became a *conventional* one with the usual quality, with no very unusual *intensity and no thrill*. The patient has done quite well on the whole, a consideration which, with the others mentioned, makes *rheumatism* the probable basis of the seagull *murmur* in this patient. The benign course makes idiopathic cystic medial necrosis unlikely.

In *some* patients (e.g., T. S. 103485) musicality may *appear* in a murmur *previously* conventionally noisy. In others (*cf.* *Figs.* 189 and 192) the musical element may disappear, leaving an aortic diastolic murmur of the usual noisy quality. *The* variations in the graphic character of musical aortic diastolic murmurs are many and include differences in the following characteristics:

1. The time interval between the beginning of the murmur and the frequency peak.

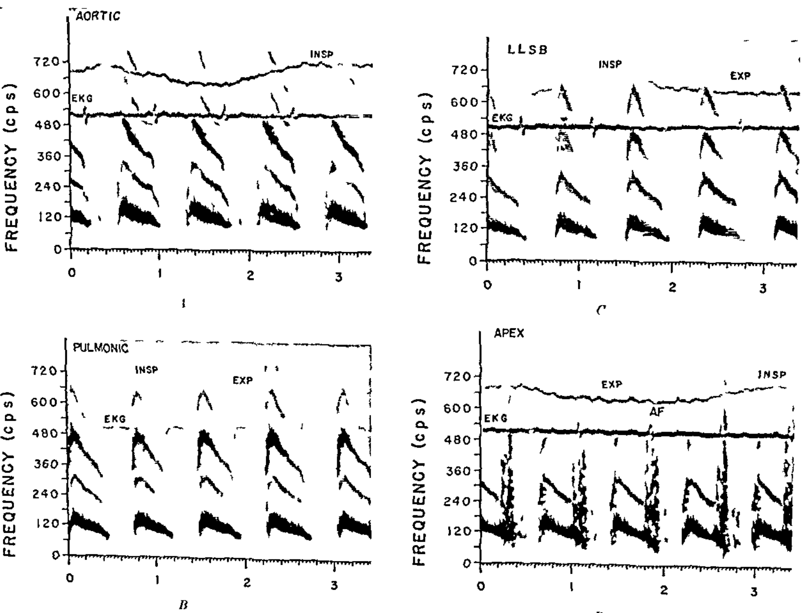


FIG. 191. Musical diastolic murmur of *retroverted aortic cusp*. In this case there is a change in slope of the harmonics with atrial systole. Rather than stopping completely they fall off more rapidly in intensity and frequency. At the apex there is a presystolic murmur which is probably of the nature of an Austin Flint. Its musicality is puzzling. Another puzzling feature is the attenuation of the second harmonic in the aortic and pulmonary areas. (In a 16 year old patient with a musical aortic diastolic murmur from *severe rheumatic fever Gelfand and Bellet* (83, *Fig.* 3) *illustrated a musical late diastolic element to the murmur.*)

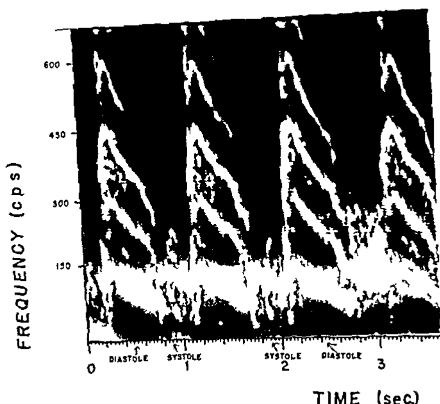


FIG 1[illegible] Retroverted aortic cusp caused by syphilis in T S (10HS5) 50 year old colored male Recorded at Erb's area Spectrogram made with phase filter (Compare with Fig 44 In this record the harmonics are not as finely demonstrated as the earlier figure because of halosis) A point of particular interest is the presence of a musical systolic murmur consisting of a single harmonic which is the continuation of the fundamental of the diastolic murmur Seemingly the anomalous valve structure was activated in systole as well as in diastole When the same patient was studied two and one half years later it was found that the aortic diastolic murmur was now of conventional noisy quality (See Fig 265)

2 The frequency level of the fundamental and other harmonics (*cf* Figures 191 and 271)

3 The frequency span between the nadir and the zenith of the harmonic

4 The presence or absence of a systolic murmur with similar harmonic constitution (see Fig 192 268 and 272)

5 The degree of cycle-to-cycle variation in the diastolic murmur (see Fig 267)

6 The presence or absence of an abrupt change in the musicality of the diastolic murmur part way through

7 The duration of the musical murmur particularly with regard to its interruption by atrial contraction (see Fig 190)

8 The presence of an increase in intensity without corresponding increase in frequency in mid-diastole with atrial contraction* or just before premature contractions (see Fig 189)

9 The alteration in tonality on transmission to the several precordial loci (see Fig 193 and 270)

10 The presence of variation with respiration—specifically an intensification early in expiration (see Fig 267)

I have studied at least one patient in whom the *mitral valve seems the likely site* for a musical diastolic murmur (see Fig 194) By means of sulfadiazine the patient was cured of subacute bacterial endocarditis in 1941 Since that time, she has displayed a musical systolic murmur at the apex (caused by mitral regurgitation) and at

* Hays and Boggan (658) picture a crescendo in a musical murmur at the time of atrial systole

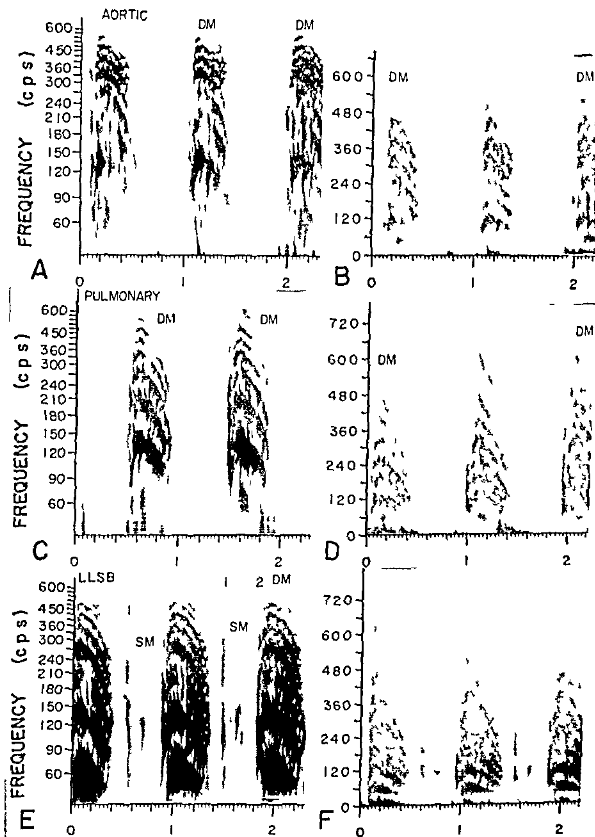


Fig. 193. Logarithmic and linear frequency displays of recordings from aortic (A, B), pulmonary (C, D) and LLSB (E, F) areas in patient with "sea gull" murmur. Note the evidences of what may be resonant absorption in a band of frequency from 150 to 300 c.p.s. (aortic area). Also note the intensification of the murmur in late diastole without rise in frequency (cf. Fig. 183).

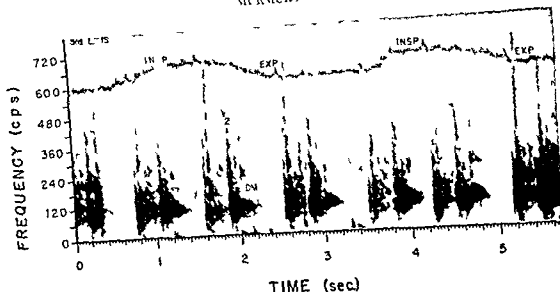


Fig 194 Calcified mitral valve

Third and fourth left intercostal spaces in L B (1,C61,) 62 year old female with advanced rheumatic heart disease and subacute bacterial endocarditis caused by *Streptococcus viridans* and cured by sulfadiazine 14 years previously There is a musical late systolic element (better shown in Fig 316) and a musical early diastolic Both are thought to have their origin at the mitral valve An opening snap was thought to be present in other recordings

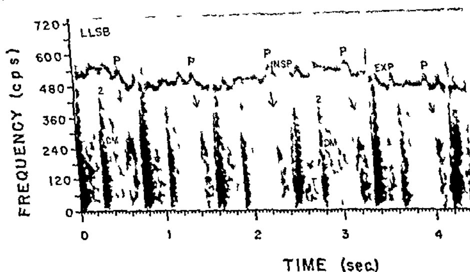


Fig 195 Musical atriosystolic sound

Recorded at LLSB two months after mitral valvulotomy for MS in D C (680606) 18 year old female The I R interval is 0.24 sec The same musical atrial murmur with the same I P interval was present four months before operation and has now been observed for a total of nine months At least two possibilities exist This may be a musical presystolic murmur or more likely it may be a musical atriopericardial murmur No particular abnormality of the pericardium over the left side of the heart was noted at operation However it is possible that contraction of the *right* atrium is responsible for the murmur The frequency of the single harmonic varied from time to time being 500 c p s in some recordings

times a musical quality to the murmur of moderate mitral stenosis Except for its musicality the murmur has the other features of a mitral stenosis murmur This patient appears then to have a musical murmur in both systole and diastole produced at the same generator In calcific aortic valve disease the distorted orifice may likewise function as a bi-directional generator of a musical

murmur in both systole and diastole. Occasionally, harmonics are seen in systole in cases of retroverted aortic cusp due to syphilis. In these instances, it is thought that the abnormal cusp prolapses into the aorta in systole, or is at least incited to vibrate during systole.

Musical presystolic murmurs are very uncommon. In the diastolic murmur of retroverted aortic

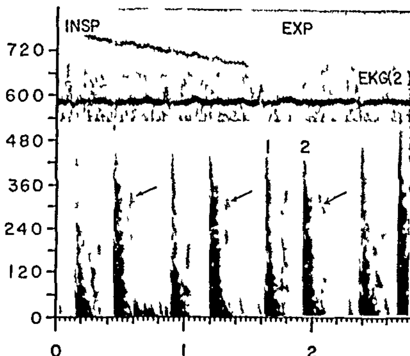


Fig. 196. Musical diastolic murmur of extracardiac origin.

This patient (A. P. 318935) had rheumatic fever in 1919 when 24 years old. The early diastolic squeak indicated by the arrows was heard on annual check ups between 1953 and 1957. There is no evidence of other cardiovascular residue of rheumatic fever. The murmur of minimal aortic regurgitation is usually high pitched but not truly musical in our experience.

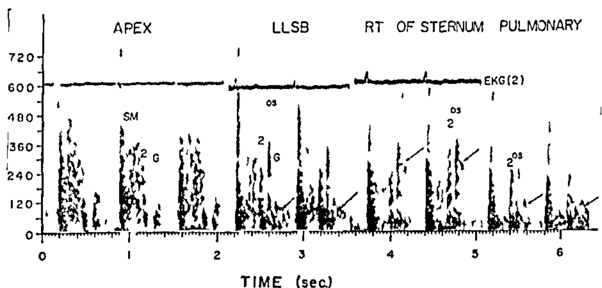


Fig. 197. Musical extracardiac early diastolic murmur (indicated by arrows) in region of lower sternum in patient (S. H. 02,689) with rheumatic trivalvular disease and mammoth cardiomegaly. The findings in the mitral area—holosystolic murmur and protodiastolic gallop—are typical of mitral regurgitation. An opening snap is present in the other three records, whether it is tricuspid or mitral cannot be said. This is probably a musical violin type of pleuropericardial rub.

Fig. 198. Musical extracardiac early diastolic murmur heard at LLSB for first ten days after valvulotomy for mitral stenosis in A. S. (66731). 38 year old woman. The mitral opening snap is evident.

cusp there may be, as noted above, a musical presystolic accentuation. Cailo (220) described a patient with syphilitic aortitis and heart failure, aortic regurgitation, a conventionally not very early diastolic murmur and in the aortic area only a presystolic "spaulement".

Figure 195 presents a most unusual case of a musical presystolic (or at least atriosystolic) murmur in mitral stenosis. It is entirely possible that this murmur was pericardial in origin. However, it was present before as well as after operation and no particular evidence of pericarditis over the left atrium was discovered at operation, nor was the anatomy of the mitral valve unusual. Lian and colleagues (944) had a case they interpreted as an extracardiac presystolic murmur but it was too evanescent to permit phonocardiographic confirmation.

On rare occasions a musical diastolic murmur may be *extracardiac in origin* (716, 942, 944). I have seen such in the first few days following cardiac surgery and in large rheumatic hearts which in addition to impinging prominently on interior thoracic wall structures may have pericardial roughening (Fig. 197). In one patient a faint early diastolic squeak, which was absolutely the only residuum of rheumatic carditis, persisted for three years (Fig. 196) but had disappeared after five years. After cardiotomy for valvulotomy or septal repair a musical diastolic murmur may be present for several days. It may be early diastolic or atriosystolic. It occurs especially often in rheumatic hearts after mitral valvulotomy (Fig. 198).

Other Musical Murmurs

The possibility of a musical cardiopulmonary murmur representing in essence a piping rale (690) was suggested by Laennec (see p. 9). It might occur in either systole or diastole. I have not convinced myself of this mechanism in any specific case. In most cases in which this is a possibility pleuropericardial rubbing seems more likely.

The musical murmurs associated with ball valve thrombus of the atrium and dissecting aneurysm of the aorta, musical venous hum, the musical uterine souffle and musical murmurs arising in narrowed arteries are of considerable interest and some practical importance. Musical murmurs of incompletely understood mechanism occur with ventricular aneurysm (p. 426).

In one patient in whom the right atrium was occupied by a ball valve thrombus[3] (J. C., J.H.H. 046029) and in a second with the left atrium as the site (H. K., J.H.H. 094258) a loud, musical systolic murmur was heard. A patient (V. S., 093271) with myxoma of the left atrium had a similar murmur. Unfortunately recordings are not available in any of these patients. From the findings of post mortem examination in the second and third and the findings of operation in the first it seems likely that tricuspid or mitral regurgitation created by the action of the ball in holding apart the margins of the valve cusps was the basis of the murmur. Immediately to mind comes an analogy to the whistle, in which a ball by rapidly cyclical occlusion of an orifice modulates a stream of air to produce a musical sound.

In the case of involvement of the right atrium there was a high pitched systolic "coo" heard at the left lower sternal border and at the very end of inspiration for the first three or four beats after the breath was held.

In the case of left atrial thrombus the murmur was likewise highly changeable. During deep expiration there was a very harsh, screeching or grunting systolic murmur immediately following the first heart sound. The murmur could be heard with the stethoscope one centimeter from the chest wall. At full inspiration this high pitched sound completely disappeared. Position did not affect the murmur, however, the musical systolic murmur completely disappeared for a few beats at full inspiration. There appeared to be an escape

[3] See ref. 1943 for a somewhat similar case.

murmur in both systole and diastole Occasionally, harmonics are seen in systole in cases of retroverted aortic cusp due to syphilis In these instances, it is thought that the abnormal cusp prolapses into the aorta in systole, or is at least incited to vibrate during systole

Musical presystolic murmurs are very uncommon In the diastolic murmur of retroverted aortic

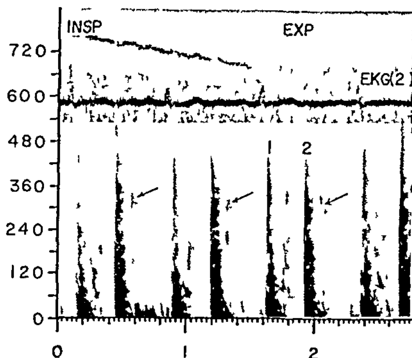


Fig 196 Musical diastolic murmur of extracardiac origin

This patient (A I 348935) had rheumatic fever in 1949 when 21 years old The early diastolic squeak indicated by the arrow was heard on annual check ups between 1953 and 1957 There is no evidence of other cardiovascular residua of rheumatic fever The murmur of minimal aortic regurgitation is usually high pitched but not truly musical in our experience

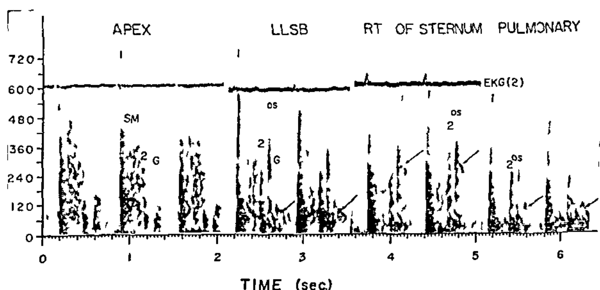


Fig 197 Musical extracardiac early diastolic murmur (indicated by arrows) in region of lower sternum in patient (S H 62,689) with rheumatic trivalvular disease and mammoth cardiomegaly The findings in the mitral area—holosystolic murmur and protodiastolic gallop—are typical of mitral regurgitation An opening snap is present in the other three records whether it is tricuspid or mitral cannot be said This is probably a musical violin type of pleuropericardial rub

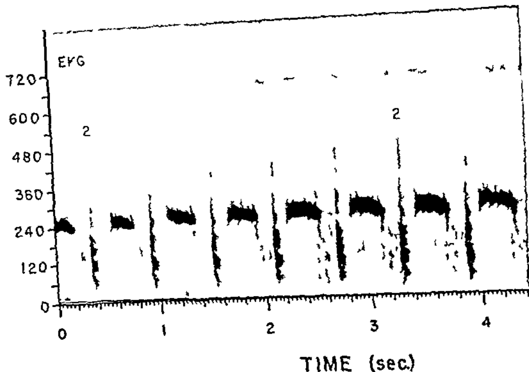


Fig. 199 Musical venous hum

Recorded at the base of the neck on the right in L. A. K. (730233) 17 year old girl with coarctation of the aorta. A musical tone occurred consistently in late diastole. S was heard in the neck with unusual clarity and was caused by hypertension. Note the gap between S and the beginning of the musical murmur; this was evidence suggesting that venous flow was responsible for the murmur, since it might be expected that a gap of approximately this duration would be necessary for opening of the tricuspid valve and build up of venous flow. For no apparent reason in the course of the recording the musicality became louder with slight rise in pitch. It also became longer by reason of beginning closer to S. There would appear to have been an acceleration of venous flow.

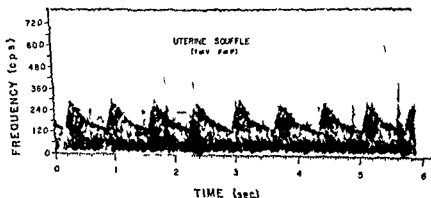


Fig. 200 Musical uterine souffle with shape of arterial pulse pressure curve. The contour of the single harmonic which is its most striking feature is determined by the *chain of proportionalities linking frequency to arterial pressure* (see text)

and this is displayed in Figure 208—a sprightly little tune is produced with at least three notes resulting from three levels of venous flow, related to atrial systole, ventricular systole and ventricular diastole.

The uterine souffle is usually noisy but occasionally may have a conspicuous musical character. (Most of the musical murmurs discussed in this section are rarer counterparts of more commonly occurring noisy murmurs.) The musical

after a few beats and with post-inspiratory apnea the murmur gradually returned, *becoming louder* and louder with each beat. It is interesting and diagnostically important that respiration had converse effects in these two cases. The usual experience is that tricuspid murmurs are accentuated by inspiration, presumably because of increased venous return to the right side of the heart. On the other hand, venous return to the left side probably falls, as a rule, with inspiration and is increased with expiration. Mitral murmurs may show variations with respiration paralleling those in venous return to the left atrium.

In the case of myxoma of the left atrium the systolic murmur was less noticeably variable. It was described as follows: *At the apex transmitted* poorly to the axilla and extending over along the left sternal border there was a loud high pitched cooing type Grade IV systolic murmur masking the first sound. Weinstein and Arata (1519) described a harsh systolic murmur in a case of myxoma of the left atrium. Block, Parker and Edwards (116, 408) described a very high pitched whistling musical systolic murmur. Caron (237) and Hoffmann (698) described a raspy systolic murmur. However in none of the literature on ball-valve thrombus of the atrium (443) have I found reference to a murmur of the type described above.

Occasionally the murmur of ruptured chordae tendineae or ruptured papillary muscle may be partly or largely musical in quality. Butterworth (203) provided me with tape recordings made on repeated occasions over a period of several months in what was probably ruptured papillary muscle from coronary artery disease. The pattern of musicality of the systolic murmur varied somewhat during the period of observation.

In *dissecting aneurysm of the aorta* a musical murmur developing abruptly over the upper thorax, usually in the first and second interspace or at the base of the neck, can be a helpful diagnostic clue. I have had experience with three varieties of musical murmur associated with dissecting aneurysm. In one group a *systolic murmur* appeared with the dissection and was explicable, seemingly, on the basis of vibrations in the lip created by the intimal rent or in the fibrous cords which usually traverse the false channel (1072, p. 52).

In another group, the murmur was likewise *systolic*, was *limited sharply to the base of the neck*, and appeared to be generated in a partially occluded innominate artery or other branch arising from the arch of the aorta (Fig. 467). In these patients the mechanism appears to be identical to that operating in other cases of musical murmurs with partially occluded arteries (see later).

In a third group, represented in my experience by a single patient, a bizarre "bull fiddle" sounding, late diastolic murmur occurred, loudest in the left first and second interspaces and audible only with longer diastolic periods such as in the compensatory pause following a premature contraction. One of the two mechanisms already *mentioned was possibly operating in this patient* whose murmur and anatomical findings are demonstrated in Fig. 9, p. 52 of reference 1071. Furthermore, the proximal diverticulum in the vicinity of the aortic valve, with accompanying lip, may have had something to do with the murmur. Finally, it is possible that the murmur may have been *generated at the aortic valve*. *Indeed* the patient had in addition a conventional noisy early diastolic decrescendo murmur. It is possible that near the end of a long diastole circumstances were such at the aortic valve that the musical murmur occurred.

Roberts (1284) described a 33 year old man with dissecting aneurysm in whom he found a very intense systolic thrill of a peculiar vibratory nature and a long coarse whistling systolic murmur over the aortic area and neck vessels. As is shown clearly in his drawing, autopsy revealed just above the right anterior coronary cusp a lip which had been dissected up in the process of burrowing back in the vicinity of the aortic ring. This lip was certainly in a position to vibrate musically during ventricular ejection and was the only structure found to account for the systolic thrill and murmur described.

Venous hums may be spontaneously musical or may become musical with a certain amount of pressure on the stethoscope or microphone. It is usually *difficult to reproduce* the musicality with any predictability. Figures 199 and 208 display two examples of musical venous hum. The loudest and highest pitched component is likely to be in diastole because venous flow is most rapid at this time in the cardiac cycle (Fig. 199). Sometimes—

uterine souffle (Fig 200) is composed of a single prominent harmonic which describes an arterial pulse pressure curve The frequency at which the generator—the tortuous dilated uterine arteries carrying an unwontedly large volume of blood because of the pregnant state of the uterus—is driven is proportional to the velocity of flow which in turn is proportional to the volume of flow which finally is proportional to pressure Thus, frequency at any one moment is proportional to pressure

Partially occluded arteries characteristically produce a noisy murmur which is related to ventricular systole with its peak in recording removed from the QRS of the simultaneously displayed electrocardiogram by an amount dependent on the distance from the heart to the obstructed vessel Occasionally the murmur may be musical and may be continuous both characteristics require special *explanation* Myers and colleagues (1136) noted that the murmur of arterial obstruction can be continuous rather than only systolic when the intra arterial pressure beyond the obstruction is so low that the pressure proximally exceeds it at all times during the cardiac cycle Musicality is not so easy to understand The mechanism is probably comparable to that in bronchial asthma (1079) That the murmur shown in Figures 201 and 202 has the contour of an arterial pressure pulse has the same explanation as was provided for the musical uterine souffle (see above) The murmur was recorded over the left carotid bifurcation in 2 men with severe generalized atherosclerosis on the basis of hypercholesterolemia

The recording in Figure 387 represents another situation in which there may occur a musical murmur with distinct analogies to uterine souffle and which represent in effect relative stenosis of arteries The patient has tetralogy of Fallot with pulmonary atresia This is so-called pseudotruncus arteriosus since the blood supply of the lungs is from the aorta via bronchial arteries The low pressure in the system which these arteries supply accounts adequately for the continuous nature of the murmur and an asthma-like *mechanism* for the fact that the contour of the *fundamental* is that of an arterial pulse pressure curve

It will be clear from the above discussion that hemodynamic factors are clearly reflected in the shape of the harmonics of musical murmurs This *fact is illustrated* by the murmur of calcific aortic stenosis retroverted aortic cusp and by venous hum and the various vascular murmurs with the pattern of an arterial pulse pressure curve

EXTRACARDIAC MURMURS

Pericardial Friction Rubs

Pericardial friction rubs (Fig 203) are really murmurs usually of the noisy variety occurring usually but not necessarily invariably in both

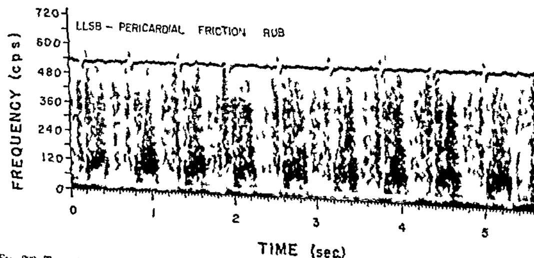


Fig 203 Typical pericardial friction rub in a patient with acute tuberculous pericarditis *Note the rather diffuse frequency composition* Accentuation of the friction sound *in ventricular systole early diastole and atrial systole* is evident

Fig 201 Musical murmur of partial arterial occlusion

Recorded over left carotid bifurcation in J I P (184964) 49 year old male with idiopathic hypercholesterolemia There is a noisy murmur with its peak at about the same time as the second heart sound which is faintly visible (2) There is a continuous pure tone with changeable frequency level describing an arterial pulse pressure curve Continuous murmurs like that shown here and in the next figure are important in the early diagnosis of carotid artery insufficiency (317 1)

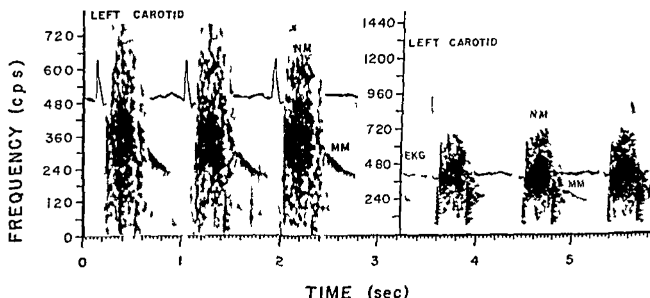


Fig 202 Partial arterial occlusion

Recorded over left carotid artery of C L (781887) 62 year old man with severe generalized arteriosclerosis Displayed on 720 and 1440 c p s scales The murmur at the peak of systolic flow (N M) is largely noisy (with submerged harmonics) It is followed by a musical murmur

Extracardiac murmurs which appear to have their origin in pericardial roughening are discussed on pp 138 and 206 *Bourne* (141) described an ostensibly normal recruit who displayed a baffling *murmur which corresponded with all of the heart's movements*—systolic and diastolic of the ventricles and systole of the auricles—producing a triple "baffle sound" (It was eliminated by inspiration and was only heard during the last *half of expiration and the beginning of inspiration*.) This was presumably a pleuropericardial friction which *happened* to be detected at the time of the examination of a person with subclinical pleurisy However it is possible that the *phenomenon* was of long standing character It is a pity that circumstances did not permit follow up

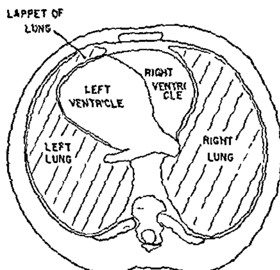


FIG 205 The postulated mechanism of the cardiopulmonary murmur

Cardiopulmonary Murmurs

As Laennec pointed out the movement of air in lung tissue through pressure by the contracting, less likely by the dilating heart on a lappet of lung is a possible origin of some murmurs (Fig 205) It is to these that the term "cardiopulmonary" is most legitimately assigned Often it is difficult to categorize a murmur any further than extracardiac and the differentiation of pericardial (or pleuropericardial) and cardiopulmonary origin is impossible See page 15 for Hope's entertaining description of cardiopulmonary murmurs in two young men who wore tight waistcoats

Characteristically cardiopulmonary murmurs vary with respiration Some are louder or present only in inspiration (the *inspiration à rythm systolique* of Tripier and Devic (1470)) some in expiration The murmur is usually systolic In fact I have not encountered a diastolic murmur I could feel confident was cardiopulmonary (51 p181) although diastolic murmurs which appear to be of pleuropericardial origin are familiar from experience in several cases The part of systole

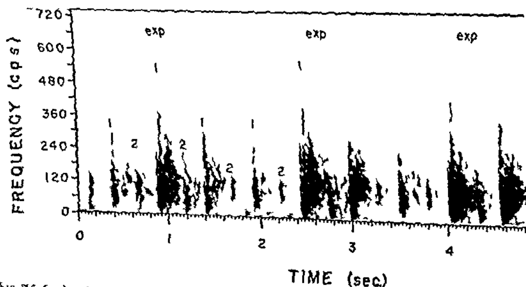


FIG 206 Cardiopulmonary murmur at apex in patient with marked ascites There is a *decrescendo* systolic murmur present only in *expiration*—probably an exaggerated vesicular breath sound (see p 478)

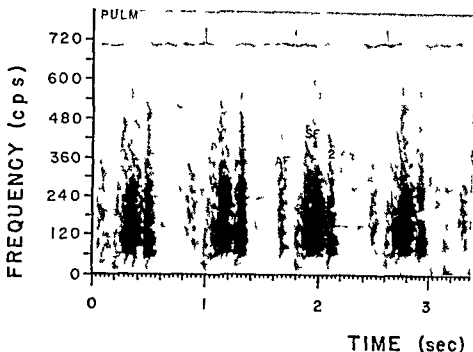


FIG 204 Atrial friction and circumscribed systolic friction in I. M. (618231) 62 year old female with recurrent pericarditis of obscure cause. Background noise throughout record.

systole and diastole, and having a characteristic superficial quality variously described as leathery, scratchy, etc. Pericardial friction rubs are notorious for their variability of location and intensity from time to time. There is no usual place of maximum audibility. In fact, when pericarditis is suspected it is important to listen frequently in all parts of the precordium and with the patient in various positions.

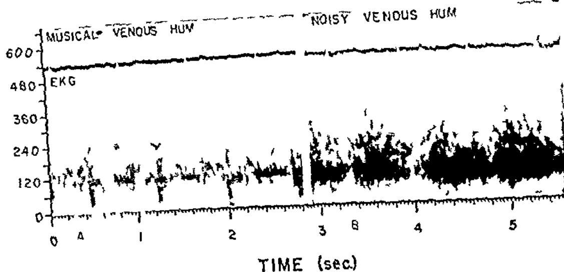
Pericardial friction rubs may, during their evolution or devolution, be limited to systole or much more rarely to diastole. In full blown form, they have three components (301): systolic, protodiastolic, and presystolic (atrial). This may give a railroad train "choo-choo-choo" rhythm to the sound.

At times a pericardial friction rub may be mistaken for the systolic and diastolic murmurs of aortic valve disease. Pressure on the precordium with the stethoscope is a time honored method for accentuating a pericardial friction sound and differentiating it from an endocardial murmur. The mechanism of the accentuation is probably acoustic filtration, although closer apposition of the rubbing surfaces may play a role in the phenomenon, especially in children with pliant chest walls. Ribaud *et al* (1268) questioned the specificity of this sign and pointed out that endocardial murmurs may be accentuated by the same maneuver.

In the oscillogram there is little or no basis for differentiating a pericardial friction rub from an endocardial murmur. In the spectrogram the quality of the pericardial friction rub is represented by the more diffuse frequency content, i.e., the more uniform distribution of energy over an appreciable range of frequency. On the other hand, endocardial murmurs as a rule tend to have a band of dominant frequency, albeit wide, with much sharper fall off above and below this level.

There is usually a silent gap between the atrial component of a pericardial friction murmur and the first heart sound (Fig 204). This feature with a PR interval of normal duration helps differentiate a presystolic friction from a presystolic murmur in, for example, patients with mitral stenosis who have had recent valvotomy.

Musical pericardial friction rubs produced by a mechanism comparable to that of the violin family of instruments have been commented on (p 206). These are frequently a long standing manifestation. Occasionally in acute pericarditis the murmur may be musical, resembling to the ear the rubbing of a wet finger on glass. Furthermore, for weeks after recovery from acute pericarditis the patient may show a grunting or croaking systolic murmur which because of its subsequent disappearance is almost certainly of pericardial origin (Fig 230).



Figs. 208 and 209. Venous Hum

Record A from the right side of the neck in a normal 23 year old female (C. F. M.) in the sitting position. The murmur at time A is musical, at times noisy. In any one cardiac cycle there are several changes in pitch of the musical murmur resulting in a sprightly little tune. When noisy the murmur has maximal intensity in diastole.

mum velocity of flow in the veins at the base of the neck. This characteristic distinguishes a venous hum (which may occur in thyrotoxicosis) from a thyroid bruit (p. 445) which is in essence an arteriovenous fistula and, as in all arteriovenous fistulas, has its maximum intensity related to ventricular systole albeit late systole. Occasionally a venous hum is interrupted and displays at least two separate components, one in systole, one in early diastole. This is most likely to occur with musical venous hums (Fig. 201) but noisy hums may also display this feature (Fig. 464). Nilson and Kramer (1145) measured inferior vena caval flow and found two maxima, one during systole and one during the first part of diastole. Atrial systole produced a reduction in the volume and velocity of flow. Holldack and Wolf (706, p. 158) illustrate a case of second degree heart block with a venous hum which was interrupted at the time of each atrial systole.

In 1937 Ivan (916) described in two patients a continuous murmur in the right interscapulovertebral space. A small tumor of the lung was present in both and responsible for the murmur in the opinion of the author. He thought, furthermore, that the murmur was of the nature of a venous hum caused by compression of pulmonary veins.

By older writers (618, p. 312) we are informed that a venous hum can be heard over the femoral vein in cases of anemia and that it is exaggerated by raising the leg. Possibly both accelerated blood flow and constriction of the veins at the inguinal ligament are factors in the latter effect.

The Carotid Bruit

The carotid bruit (referred to by Holldack and Wolf (706) as the autochthonous carotid murmur to distinguish it from the transmitted murmurs of aortic stenosis) is a poorly recognized phenomenon with practical significance. Laennec, Bouillaud and many others in the first 30 years or more of stethoscopy considered what we now call the venous hum to have its origin in the carotid artery, presumably through compression of the artery by skeletal muscles in its vicinity. The protagonists for the venous origin of this sound did such a thorough job that all ideas of the origin of murmurs in the carotids were discarded. Although compression by surrounding muscles is unlikely, there are several circumstances in children in particular which conspire to result in a murmur at the base of the neck. Firstly, the bifurcation of the innominate artery into the right carotid and right subclavian arteries receives a direct blast of blood from the ventricle. Secondly, there is an edge at this point which splits the stream of blood. In children with familiarly

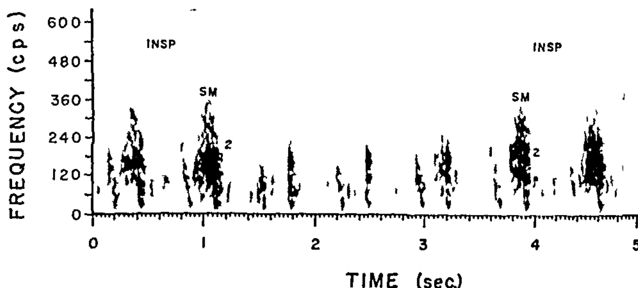


Fig 207 Cardiopulmonary murmur at apex in patient (P R 364090) with flat chest and cardiomegaly caused by hypertension A crescendo murmur occurs in systole Mechanism probably as in last figure

occupied by the murmur varies For example, in Figures 206 and 207 are presented two instances of cardiopulmonary murmur one is decrescendo and early systolic occurring mainly in expiration, the other is crescendo and late systolic occurring mainly in inspiration

Thayer (1469) claimed that these murmurs may be heard with great intensity in the back over the left lung

It is theoretically possible for a cardiopulmonary murmur to be musical as a result of the movement of air through bronchioles narrowed by some other mechanism the whole situation being comparable to that in bronchial asthma Calo (218) described the case of a chronic bronchitic in which he postulated this mechanism in this case furthermore the murmur was exceedingly intense and was diastolic in timing Luisada and colleagues (944) applied the same explanation for certain systolic and diastolic musical murmurs Usually a pleuropericardial violin like mechanism for musical extracardiac murmurs seems on the basis of circumstantial evidence more likely than a *cardiopulmonary origin*

VASCULAR MURMURS

Thoracic Arteriovenous Fistulas

Thoracic arteriovenous fistulas will be discussed in connection with the conditions simulating patent ductus arteriosus (p 40?)

Venous Hum

Venous hum (838) is because of its frequency and because of its possible confusion *with lesions* of grave significance one of the major auscultatory phenomena (Figs 208 209) It is usually maximally audible at the base of the neck, especially at the right It may be audible in fact loud over the upper part of the chest in young children suggesting some lesion of grave significance For *example in fourteen children of ages 3 to 14* years cardiac consultation was sought because of a mistaken diagnosis of patent ductus in three and of aortic regurgitation in two (1178) The abrupt enlargement of the veins with confluence of several streams may be responsible for the usual localization of venous hum It occurs most often and in most striking form in children in the upright position and in inspiration—all conditions associated with increase in velocity of flow In girls it usually persists until later in life than in boys A venous hum can often be elicited by having the subject turn his head away from the side of auscultation thereby putting the veins on the stretch or otherwise narrowing them and can be abolished by light pressure on the veins above the point of auscultation It is more frequent in *anemia the reduced viscosity of blood* is probably responsible Like most murmurs it is exaggerated by fever It also may be increased by thyrotoxicosis Venous hum may occur more frequently with coarctation of the aorta possibly because of increased flow in the part of the body proximal to the coarctation

The venous hum is most often a continuous murmur which has its maximum intensity in ventricular *diastole* (Fig 209) at the time of maxi

the neck on the right may occasion concern about the possibility of mild aortic stenosis (Fig 210) If the murmur is heard only at the base of the neck and not in the aortic area it is probably a *carotid bruit* and of no serious prognostic significance

The carotid bruit occurs in association with aortic regurgitation (Fig 211) because of the rapid ejection of a large stroke volume A systolic murmur at the base of the neck on the right need not indicate associated aortic stenosis

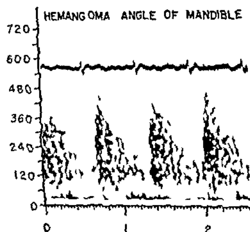


Fig 1 Congenital AV fistula
Recorded at angle of mandible on left in J T (301 440) 40 year old female

Peripheral Vascular Murmurs

Peripheral vascular murmurs (407 703) include, of course the murmur of arteriovenous fistulas and other fistulous lesions (406) of partial arterial occlusion, and of arterial collaterals Certain special categories such as uterine souffle, mammary souffle the Cruveilhier Baumgarten murmur and cephalic bruit will be discussed separately

In arteriovenous fistulas (Figs 212 to 214) the murmur is characteristically continuous with its peak in late systole and delayed by an interval dependent on the distance of the fistula from the heart The intensity of the murmur can be reduced by pressure on either the afferent artery or the efferent veins although pressure on the former is more effective in obliterating the murmur Slowing of the heart (Branham's sign) usually accompanies obliteration of the fistula The murmur is essentially the same whether the fistula is of traumatic origin (Fig 215) represents a congenital malformation (Fig 212 to 214) or is a functional AV fistula as in a toxic goiter (Fig 216), a highly vascular neoplasm such as the metastases of chorionepithelioma (Fig 217) the bone lesions of Paget's disease a pregnant or myomatous uterus or even lactating breasts

Bonan and Calo (127) found the typical murmur in an arteriovenous fistula of the forehead

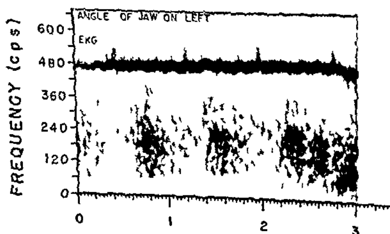


Fig 13 Congenital AV fistula at angle of jaw on left in R B (20424) 53 year old white male An incomplete operation was performed seven years previously There is a cystic area in the mandible Although the murmur is not ... its top has the shape of an arterial pulse pressure curve

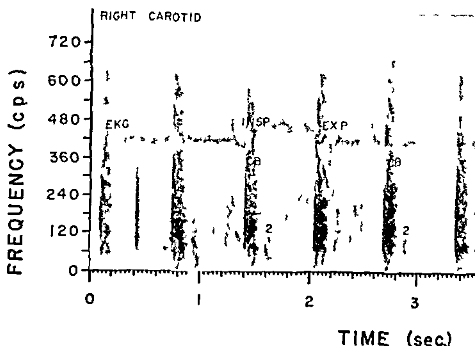


Fig 210 Carotid bruit

Base of neck on right in B A J (A68968) 14 year old female who had acute rheumatic fever at the age of 5 years There is no definite evidence of residual valvular damage The circumscribed sound in the neck probably is generated locally in the innominate carotid subclavian axis since its onset is appropriately late and there is no murmur in the aortic area At the apex there was a telesystolic click probably caused by pericardial adhesion

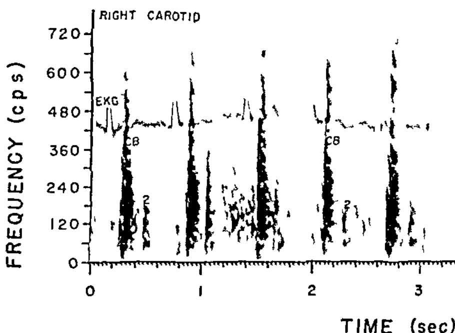


Fig 211 Carotid bruit

P S (247595) 32 year old female has aortic regurgitation but no impressive murmur of aortic stenosis in the aortic area

vigorous circulations circumstances for the production of a murmur at the innominate carotid subclavian junction are likely to exist at the peak of systole (Fig 210) and to be favored further if fever and anemia (706, p 151) are present In children who are being followed for evidence of cardiac involvement after acute rheumatic fever the presence of a systolic murmur at the base of

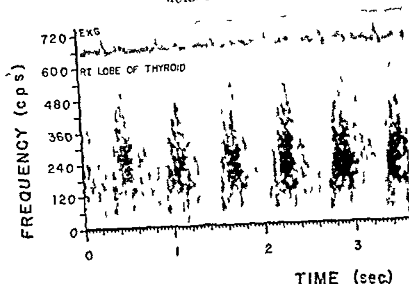


FIG 216 Thyroid bruit in E D (3?3669) 46 year old female The murmur is maximal in systole unlike a venous hum which is usually maximal in diastole See Figure 460 for the Lerman Means scratch in this patient

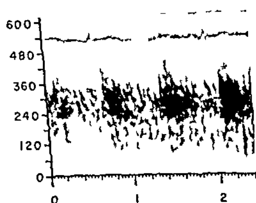


FIG 217 A Arterial souffle

Murmur produced in vascular metastases of chorionepithelioma in retroperitoneal area of upper abdomen and audible over most of the abdomen and lower back At operation uncontrollable bleeding was encountered and the patient (P G ??? 26 years old) died The top of the murmur has the shape of an arterial pulse pressure curve

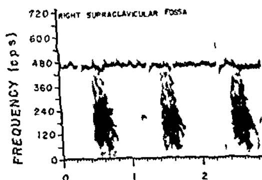


FIG 217B Atherosclerotic stenosis of right subclavian artery

Recorded in right supraclavicular fossa of I L ((?63?04) 2? year old male with severe hypertension and advanced premature atherosclerosis The murmur is distinctly musical After the application of heat to the right arm (shown here) the murmur became longer extending somewhat into diastole The *principle established* by Myers and colleagues (1136) is well illustrated

incident to bleeding with leeches Figure 217 presents a case of arteriovenous fistula at the same site secondary to trauma with skull fracture

Malmer (1026) described a 12 year old boy with a hemangioma in the left upper *quadrant of* the anterior abdominal wall producing a murmur which was well heard over the heart and led to a mistaken diagnosis of congenital heart disease Firm pressure on the tumor which felt like a bag of worms abolished the murmur

Renal arteriovenous fistula is a rare occurrence has in all reported instances been accompanied by a loud continuous murmur (627A) A congenital *aneurysm* with subsequent rupture may be the mechanism in some cases Hypertension is usually present and combined with a murmur in the proper site makes the diagnosis

In Paget's disease of bone vascular lesions functionally equivalent to AV fistulas develop in the involved areas of the skeleton The cardiac *output* may be raised and the oxygen content of

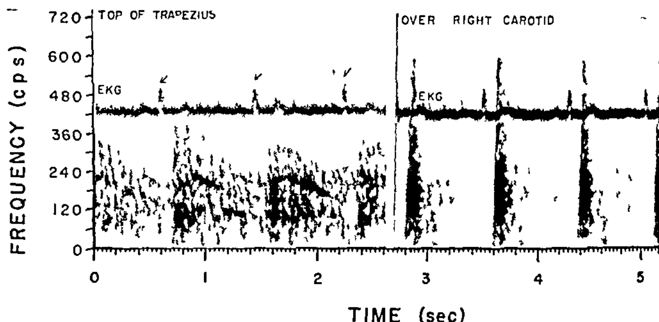


Fig 214 Systemic pulmonary fistula

A I B (762109) 31 year old male had a right upper lobe lesion previously interpreted as tuberculous A continuous murmur with high pitched musical quality was present in the right supraclavicular fossa and could not be obliterated by pressure B P was 110/60 mm Hg X ray of the chest showed abnormally large vessels extending from the right hilum to a lobulated lesion located at the extreme right apex behind the clavicle The recording over the ridge of the trapezius (*left*) showed a continuous murmur with the general pattern seen in peripheral AV fistulas but with a rather unusual harmonic pattern especially as regard the harmonic occurring in diastole and located at a frequency of about 600 c p s Over the right carotid (*right*) there was an impact type of sound suggesting high flow in that area the sound had the appearance of a carotid bruit (*cf* 210 and 211)

At operation both the supraclavicular fossa and the thorax were explored The first intercostal artery was several times normal size and there appeared to be communications between it (as well as small branches of the right subclavian artery) and the pulmonary vasculature in the right upper lobe These communications were severed After operation the murmur was no longer present

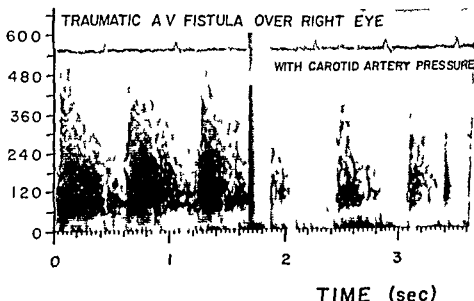


Fig 215 Traumatic AV fistula

Recorded over right eye in I G (451462) 37 year old male with an AV fistula in the right frontal area following skull fracture The intensity of the murmur is greatly reduced by pressure on the right carotid Note that in the record on the left the continuous murmur has an abrupt marked accentuation at a distance after the QRS consistent with the time necessary for transmission of the arterial pulse from the heart and that the top of the murmur has roughly the shape of an arterial pulse pressure curve

Broeklank suggested that the murmur is especially likely to develop if cervical rib is present

In coarctation of the aorta there may be a striking murmur often continuous over the various collaterals

Uterine Souffle

The uterine souffle is of arterial origin that is it arises in the uterine arteries which are called on to carry a much increased volume of blood because of the pregnant state of the uterus. The arterial nature of this murmur is proved by the shape of the fundamental in the case of the musical uterine souffle (Fig 200) and by the fact that there is an appropriate delay after the first sound in the case of the noisy uterine souffle. Burwell (202) points out the functional similarity between the placenta and an AV fistula. The identity of the uterine souffle and the murmur of AV fistula strengthens the analogy.

The blood flow to the uterus in pregnancy is about 500 cc min on the average (202A). Relatively high pressure and oxygen content of uterine venous blood support the view that the placenta is a low resistance area.

The uterine souffle is usually heard over one or the other side of the abdomen in the late stages of pregnancy. It is a phenomenon which is by no means constant but which tends to come and go for no apparent reason. Position of the fetus with pressure on or other distortion of the uterine arteries may be a factor. Uterine souffle is not a specific sign of pregnancy since it may occur in association with a large myomatous uterus or large ovarian tumor. The fact that the rate of the uterine souffle corresponds to the maternal heart rate facilitates differentiation from the funic souffle and the murmur of fetal cardiac malformation, the rate in the latter two instances is that of the fetal heart.

Mammary Souffle

Late in pregnancy and during the early partum ent period one may hear over the upper margin of either breast or usually both a murmur which full blown is continuous (Fig 218) with late systolic accentuation like the machinery murmur of patent ductus arteriosus. When present in less than full blown form it can be recognized by ear (and demonstrated by recording) that the mur

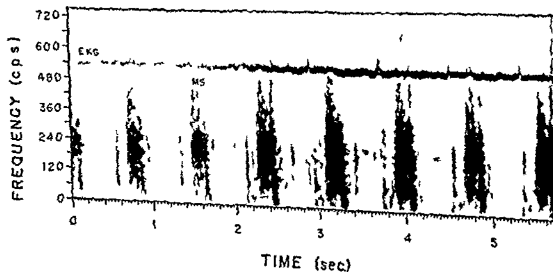


Fig 218 Mammary souffle recorded over the upper margin of the breast early post partum in L. C. (623309). The murmur in this recording is long or late systole and extended across the second sound. The findings was consistent with an arterial origin. During the recording when pressure was applied to the microphone there was essentially complete obliteration of the murmur. On release of pressure the murmur became almost continuous extending first into late diastole and resembling the murmur of PDA. Although this phenomenon occurred in full blown form in a relatively small percentage of cases we have observed at least two others (N. S. C. ... and I. C. ...) with striking changes as those shown here.

venous blood returning from an affected limb is increased as in any AV fistula. Furthermore, not only is there increased heat over the involved areas, but also a bruit may be audible, for example, over the skull and tibia. When in the skull the murmur may be troublesomely audible to the patient. Poppen (1217) suggested that narrowing of foramina at the base of the skull *with constriction* of arteries might be the mechanism.

A murmur may be heard over the spleen when *it is enlarged from any cause (107A). The murmur* is likely to have an arterial pattern with a peak in late systole; occasionally it is continuous. A *syndrome of splenic arteriovenous fistula with* ascites and the characteristic continuous murmur over the lower posterior portion of the left rib cage has been described *(241C). Because* of the significant occurrence of aneurysm formation of the splenic artery with rupture during pregnancy, the occurrence of this syndrome in the puerperium is probably of note.

Cavernous hemangioma of the liver, a vascular hamartoma, occurs somewhat more often in the left lobe and in females (1387A). A murmur, either systolic or continuous, and sometimes with thrill, may be an important clue to its presence. The lesion is frequently amenable to resection.

Partial occlusion of a peripheral artery produces a systolic murmur with the Christmas tree configuration of an ejection stenosis murmur. The peak of the murmur appears to coincide with the peak of the arterial pressure pulse. Occasionally, the murmur may be continuous in accordance with an important principle which was first elucidated by Myers and colleagues (1136) and to which I have had occasion to refer already. When the obstructive disease is sufficiently severe or collateral circulation is inadequate or pressure distal to the obstruction is lowered through vasodilatation the murmur may be continuous simply because pressure proximal to the obstruction is at all times sufficiently higher than that distal to the obstruction to produce a murmur. Myers and his colleagues showed that a systolic murmur of partial obstruction of the artery to a limb could be converted into a continuous one by having the subject exercise that limb. The interpretation was that vasodilatation instigated by exercise resulted in a drop in pressure distal to the obstruction—the condition necessary for a continuous murmur.

The value of auscultation over peripheral vessels in cases of suspected or proved peripheral vascular disease *cannot be overemphasized (1448)*. In atherosclerosis, narrowing of the ostium of the left subclavian with systolic murmur in the left *supraclavicular fossa and reduced blood pressure* in the left arm is fairly frequent (922).

In the aortic arch syndromes (1312)—narrowing *or obliteration of the mouths of the great vessels* arising from the arch of the aorta—a continuous murmur simulating that of patent ductus arteriosus may be heard in the vicinity of the clavicle, especially in cases of almost total obstruction of all branches at the arch. The mechanism is clearly that elucidated by Myers and his colleague (1136). A systolic murmur from lesser grade of ostial obstruction occurs rather frequently (837).

Hinohara (685) of Kyoto devised a method for recording sounds from the esophagus. He concluded that the method has particular virtue for recording from the vicinity of the aortic arch. In a patient with the young female arteritis variety of the aortic arch syndrome (which seems to occur unusually frequently in Japan) he recorded a continuous murmur from the esophagus in the vicinity of the arch. Presumably the continuous murmur could not be detected on the surface of the chest.

Edwards and Levine (405) point out that a systolic murmur heard in the supraclavicular fossa or axilla with the arm in certain position can be a useful clue to the diagnosis of thoracic outlet syndrome. Compression of the subclavian artery is the mechanism. In the thoracic outlet syndrome arterial obstruction may be progressive because of the secondary atherosclerosis which is incited at the site of repeated trauma to the artery.

In the 1920 edition of his small book on heart disease Brockbank (182) wrote as follows (p. 97):

> A very well marked systolic murmur simulating exactly an aortic murmur is sometimes heard over the manubrium sterni and base of the heart in quite healthy persons who are holding the shoulders well back. . . . It may be heard when the subject being examined is holding his vest well up for auscultation at the base of the heart. It disappears at once if the shoulders are brought forward to remove the pressure.

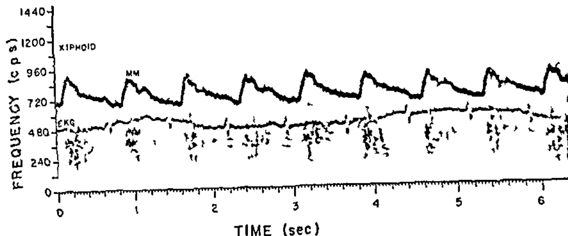


FIG 220 Musical Cruveilhier Baumgarten murmur of arterial type

In B S a 40 year old man with Laennec's cirrhosis a dilated tortuous vessel seemed to *perforate to a subcu*taneous location in the angle between the xiphoid and the left costal margin It coursed cephalad along the left sternal border and appeared to perforate into the anterior mediastinum at the level of the fifth intercostal space The vessel was thin walled like a vein but had an arterial pulse Over it was a musical murmur (MM) with the appearance shown here It was made up of a continuous pure tone (a single harmonic or fundamental) with the shape of an arterial pulse pressure curve It was located at a higher frequency level than that of most musical arterial murmurs (see Figs 201 and 202) There was an intermittent *noisy murmur* (NM) at a lower frequency level As in other recordings the rise and fall of the EKG indicated inspiration and expiration respectively There was no definite change in the murmur with respiration or with the *Valsalva maneuver* *Localized* pressure at either the caudal or the cephalad point of perforation obliterated the murmur The murmur was audible *for* a distance of only about two inches from the anomalous vessel

A more conventional venous hum had been present for at least two months before the appearance of this dilated vessel The arterialized nature of the blood was *indicated by* an oxygen content of 96 per cent Increased hepatic arterial blood flow in alcoholic cirrhosis *opening of anastomoses* between hepatic arterial radicles and portal vein radicles and indirectly the development of communications between the hepatic artery and porto caval venous collaterals were suggested In this case blood was thought *to be sluiced fairly* directly from a branch of the hepatic artery to a venous collateral draining into the caval system In essence there was an arteriovenous fistula

It should be stated that the man had had *splenectomy* previously It is my opinion that the anomalous vessel and its murmur were not related to this procedure

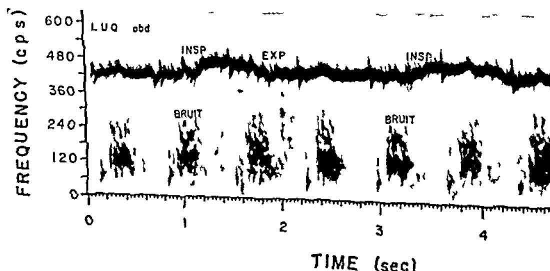


FIG 221 Hepatic bruit associated with massive liver metastases

I B (F125b) 51 year old male had massive liver *metastases* from an adenocarcinoma of the sigmoid colon The murmur over *the left lobe* of the liver begins about 0 12 sec after the first sound Since it occurs with the peak of arterial flow it is probably arterial in origin

mur begins rather abruptly in late systole, as judged by its relation to the heart sounds, and *extends slightly into early diastole* The murmur is fickle in its intensity and has a rather superficial quality Slight pressure on the stethoscope may change its quality and confer upon it partial musicality It can be obliterated entirely by moderately firm pressure with the stethoscope (Fig 218 *B*) although a lesser degree of pressure may exaggerate it I believe it is of arterial origin It bears, therefore, some interesting similarities to uterine souffle which likewise is produced through the increased demand of an organ for blood supply resulting from augmentation of a physiologic function The observations (553)(733) that a striking venous pattern over the breasts is demonstrable by infrared photographs in these patients and that the murmur can be obliterated by pressing with the edge of the hand at a point between the stethoscope and the main part of the breast are not inconsistent with an arterial origin of the murmur With any hyperemia of the breast a prominent venous pattern is to be expected Pressure distal to the stethoscope can be expected to interfere with arterial flow

Obviously, the practical importance of the phenomenon resides in the very real risk of confusing the murmur with one of cardiac origin In some cases a congenital heart lesion has been suspected and special diagnostic studies performed Like so many *other situations in medicine in general and in connection with* cardiovascular sound specifically knowledge that the phenomenon may occur is the main insurance against diagnostic error

The mammary souffle has been referred to by Gilston and McPhaul (553), Grant (584), Bonham Carter and Walker (132), Scott and Murphy (1371), and Jones (775) the last writer emphasized that it may be a musical murmur like the uterine souffle, which it resembles also in variability and mechanism In 1949 Wells and colleagues (1529 Fig 31) described what they called a mediastinal hum in a 24 year old, seemingly normal, pregnant woman The murmur was heard over the lower central sternum and following delivery [the] murmur was less intense and rather more continuous ' This may have been a mammary souffle

The Cruveilhier Baumgarten Murmur

The Cruveilhier Baumgarten murmur (946) is heard over the venous collaterals, connecting the portal and caval venous systems on the abdominal wall Laennec's cirrhosis of the liver is the most frequent cause of the portal hypertension which results in development of these collaterals—

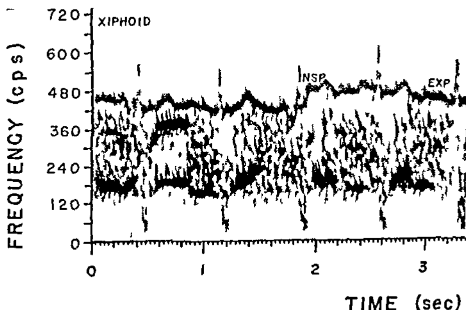


Fig 219 Cruveilhier Baumgarten murmur in V B (765876) 66 year old woman with primary biliary cirrhosis The murmur was well localized to the xiphoid and often exclusively diastolic being easily confused for an aortic diastolic murmur In this recording it is continuous and has a musical quality

side of the hand on the abdominal wall between the site of auscultation and the umbilicus.

Herrick (674A), Dock (360A) and others have proposed that there is a relative and probably an absolute increase in hepatic arterial *blood flow* in Laennec's cirrhosis; that there are abnormally wide arterioportal anastomoses constituting in effect arteriovenous fistulas, and that these contribute to the portal hypertension. It is entirely possible that the murmur recorded over the liver at laparotomy is generated in these communications. It is less likely that the murmur recorded at the skin surface is of this origin. However the case shown in Figure 220 would support the notion that the murmur is generated in arteriovenous fistulae. The continuous murmur is musical with a harmonic which describes an arterial pulse pressure curve. The murmur was well localized to the angle between the xiphoid and left costal margin where a tortuous dilated vein-like structure displayed an arterial pulsation. The *murmur is* thought to be the result of abnormal communications from the hepatic artery to the portal vein and eventually to these collateral venous channels according to the mechanism discussed by Herrick and Dock.

McFadzean and Gray (1064) of Hongkong have studied a case with a murmur of the type shown in Figure 220 and provided convincing evidence that it is generated in hepato-porto-systemic arteriovenous fistulae which develop by the mechanism *of* Herrick and Dock. The murmur was continuous with systolic accentuation. At laparotomy the murmur could be obliterated by *occluding* the hepatic artery. Occlusion of the portal vein had *no effect* on the murmur. Lipiodal arteriograms of the liver obtained at autopsy in the same case demonstrated passage of the contrast *medium* from the hepatic artery into large branches of the portal vein. Although they termed *the murmur* a 'venous hum' in their title they concluded that it differed in many respects *from that described* [by others] and probably had its origin *in arteriovenous* shunts in the liver.

Let no one think that the differentiation from cardiac murmurs is always obvious. Gwyn (621) described cases in which the continuous murmur was limited to the precordial area and was taken to indicate *congenital* heart disease. He suggested with some basis in anatomical observation that in many of these cases a sizeable venous sinus lies immediately beneath the lower sternum. Subcutaneous collaterals often penetrate in the vicinity of the xiphosternal joint. Localized pressure with the finger tips at the point of penetration may obliterate the murmur.

Cephalic Bruits

By a strange human frailty, auscultation of the skull seems to be the one thing most likely to be neglected in a routine neurological examination. Cephalic auscultation is a forgotten practice and even when the patient calls attention to the fact that he hears noises in his head they are so likely to be ascribed to some form of auditory hallucination or to tinnitus that the examiner rarely thinks of checking the patient's statement with a stethoscope.

Cushing and Bailey, 1928 (321A)

Cephalic bruits (1009, 1169) have long been recognized (see Fisher on p. 17) but recently have enjoyed a well deserved revival of interest. They are a heterogeneous class comprising murmurs generated in arteriovenous fistulas (Fig. 223), some of which are probably venous hums produced in the large sinuses, others generated in arteries narrowed for one reason or another. *Ocular bruits* (277), murmurs heard over the orbit, are a subcategory. Cephalic bruits are among the dramatic aspects of medicine, in this respect rivaling the very loud heart *murmurs*, especially that of retroverted aortic cusp (p. 269). The interesting anecdotes concerning *noises heard* by the patient himself, *diagnoses* made over the telephone (627) etc., are similarly *legion* in these two types of case. Ocular bruits are detected by applying the bell of the stethoscope *to the upper* lid of the closed *eye*. *Cephalic bruits are often* especially disturbing to patients because of the *peculiar noise*. *The complaint of* 'roaring in the head' or of the hearing of any other variety of unusual noise should prompt auscultation of the cranium, including the eyeball, before the symptom is *interpreted as* tinnitus or worse, psychoneurosis.

Disappearance with carotid occlusion is a helpful indicator that the sound is of vascular origin and generated above the neck (1217). It is pos*sible, of course,* for the murmur to continue because the vertebral artery has not been *occluded*

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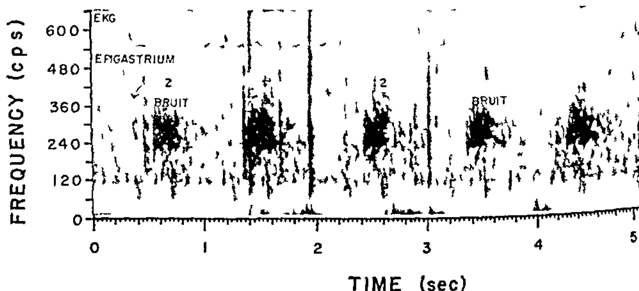


Fig 222 Arterial souffle
Recorded in epigastric area of 34 year old male (G F 557871) with Hodgkins disease for which a great deal of x ray therapy had been given to the upper abdomen The spleen was little enlarged The murmur was thought to be produced in an artery compressed by post radiation scarring The sound is concentrated at a frequency of about 300 c p s and has the appearance of the top cut off an arterial pulse pressure curve Background noise

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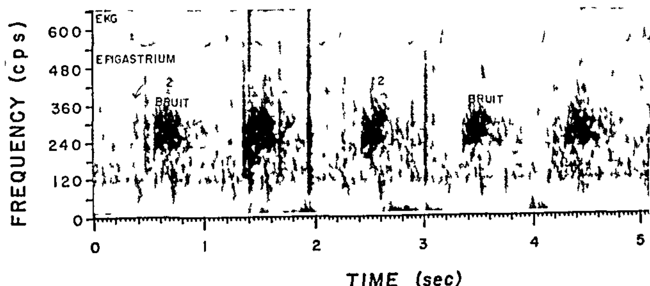


Fig. 222. Arterial souffle.

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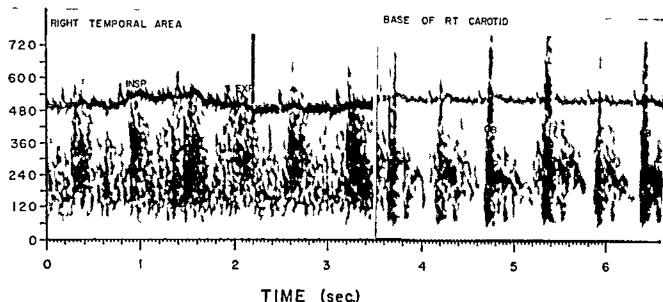


FIG 223 *Cephalic bruit* recorded in the right temporal area in V M (B18376) 18 month old child. The continuous murmur has a late systolic accentuation characteristic of arteriovenous fistula or at least indicative of arterial origin. An origin in the venous sinuses, for example, is unlikely. A congenital vascular malformation appears to be present. *Carotid bruit* and probable musical venous hum in the same patient. Recorded at the base of the neck on the right. About 0.12 sec after the QRS there is a rather long transient. It is followed by a single harmonic and there is an inconstant harmonic in diastole. The second sound is in a normal position. The transient is probably produced at the bifurcation of the innominate. The musical elements may be of venous origin. With arteriovenous fistula in the head, carotid bruit and venous hum are more likely to occur.

An interesting phenomenon is accentuation of the bruit of an intracranial lesion when pressure is applied to the *contralateral* carotid artery. Apparently the maneuver results in increased flow through the area where the murmur is generated. The possibility of a transmitted cardiac murmur must always be kept in mind and excluded by precordial auscultation. The neurologist must keep other systemic disease in mind in this as in other situations and must remember that fever or anemia may be responsible for cephalic bruits. In our present state of knowledge cephalic bruits in children are always of more doubtful significance than those in adults. Poppen (1217) states that a bruit is very frequently present over the mastoid process of children. He thought it to be generated at the point where a vessel penetrates the skull and claimed it could be obliterated by local pressure. In hemiplegic patients or patients with neurologic manifestations which may be on a vascular basis, auscultation of the head is indicated, although the clinician must guard against being confused thereby and uncovering more "red herrings" than worthwhile information. He must be prepared to be unable to interpret the findings.

In Hamburger's classical paper (627) one patient, Mrs McC, still had an unexplained cephalic bruit ten years after onset and no more diagnostic features or adverse events had developed. In another patient, a Baltimore physician who died only recently at the age of 84 years, the murmur disappeared spontaneously a few months after it was first heard and some 30 years before his death.

Mackenzie (1010) described 11 patients with cephalic bruits. The responsible lesions included cerebral angioma, meningioma, carotid aneurysms (both congenital and traumatic), tumor of the glomus jugulare, incipient internal carotid occlusion, pinealoma, and cerebral thrombophlebitis.

The classical example of intracranial arteriovenous fistula with murmur is that between the carotid artery and the cavernous sinus (1302). There is likely to be pulsating exophthalmos and the same continuous murmur which characterizes other arteriovenous fistulas. Trauma is the cause in about 70 per cent of cases, although an inherent vessel weakness may contribute. When exophthalmos is bilateral (as occurs in about 10 per

cent of cases) the side on which the murmur is louder is usually the side of the fistula. The murmur may have its onset well before the appearance of exophthalmos or congestion of the fundal vessels (1501). The loudest murmur we have heard in carotid-cavernous fistula was such a case (G T 681231). It is postulated that the communication is small in such cases with small shunt and little effect on cavernous sinus pressure but with generation of a loud murmur.

Arteriovenous fistulas may follow skull fracture just as they may occur in the thoracic wall following rib fracture. In severe anemia in patients with arteriosclerosis there may be marked murmur. Probably the arteriosclerosis need not be severe. A physician is described (23) who had two gastrointestinal hemorrhages with each of which a noise in the head was associated. Cohen and Miller (277) proved the presence of arteriosclerotic narrowing of the carotid siphon in two patients with eyeball bruit and strongly suspected it in a third. There was hemangioma in one case and profound anemia in two. A seventh case had thrombosis of the internal carotid artery. A significant point is that in six of the seven patients reported by Cohen and Miller (277) the bruit was limited to the eyeball. In applying the stethoscope to the lid over the closed eye the patient should try to relax the extra-ocular muscles as much as possible lest muscle sound conceal a vascular bruit or be mistaken for it. If the physician holds down the lid he is listening over and lets the patient keep the other eye open muscle sound may be avoided. In children a cephalic bruit may occur merely with increased intracranial pressure. Non vascular brain tumors may be accompanied by cephalic bruit sometimes perhaps through direct pressure of the tumor on vessels more often through influence of the hydrocephalus.

In some infants a physiologic *fontanel bruit* is heard over the fontanel (582). Although some have thought the fontanel bruit to be of arterial origin it seems more likely that it arises in the vein. After closure of the *fontanel* the bruit disappears in essentially all *normal* individuals. Dalgaard Nielsen (326) after surveying about 5000 individuals provided the following figures on incidence:

½ yr	[illegible]%
½–1 yr	17%
1–1½ yr	10%
1½–2 yr	7%
2–2½ yr	2%
2½ yr	0%
Adults	0%

Henoch (672) thought the fontanel bruit to be a specific sign of rickets. Delayed closure of the fontanels was the probable basis of the association.

Wadia and Monckton (1498A) have provided an extensive analysis of intracranial bruit. In a group of 228 adults who seemed to be normal they found a bruit in three. An evoked bruit that is one heard with contralateral carotid artery compression may persist into middle life. A familial aggregation of cases of intracranial bruit in normal persons was observed. The authors postulated a gene-determined variation in the pattern of the circle of Willis. Anemia and thyrotoxicosis may bring out a tendency to intracranial bruit. The pathologic bases for bruit included cerebral angioma, orbital angioma, carotico-cavernous fistula, Paget's disease of the calvarium, tumors (especially meningioma and glomus jugulare *tumor*) and atherosclerosis of the internal carotid artery especially the siphon. One should listen *for* intracranial bruit especially in cases of migraine, epilepsy or subarachnoid *hemorrhage* since angiomatous malformation may *present* in any one of these forms.

FUNCTIONAL HEART MURMURS

Men are as often deceived by their ears as by their eyes and they may *hear* ghosts as well as see them.
Latham 1847

Full of sound and fury, signifying nothing.
Shakespeare *Macbeth*

Functional murmurs represent the most difficult subject in the entire *domain* of cardiovascular sound (486 974 985A). Various names have been suggested for this category of murmur: accidental, unexplained, benign, *innocent*, cardiopulmonary, not significant, *benign* etc. Some of these terms obviously have certain special significance *and* none of them is entirely satisfactory. In a sense all murmurs are functional and benign. The murmur of atrial septal defect (see p 344) has

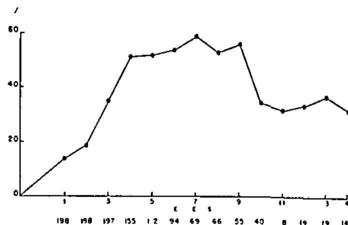


Fig 224 Age incidence of precordial systolic murmurs in normal infants and children (total of 1 264 patient years) (Courtesy of Epstein (431) and *Journal of Pediatrics*)

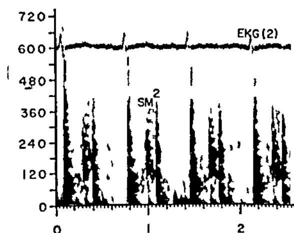


Fig 225 Extracardiac murmur

Circumscribed late systolic murmur in patient V H (A81746) 11 years old with history of rheumatic fever two years previously A persistent apical systolic murmur had been occasion for concern and had been interpreted as indicative of mitral regurgitation Because of its circumscribed character and abrupt onset extracardiac origin is likely

its auscultatory features and mechanism in common with some functional murmurs yet cannot be considered innocent Some of the murmurs which accompany rheumatic carditis severe chronic anemia, vitamin deficiency, heart failure dilatation of the heart thyrotoxicosis, and so on, are also functional, but by no means innocent Review of the literature leads to the conclusion that all loud systolic murmurs and all diastolic murmurs regardless of intensity are usually considered 'organic', all *systolic murmurs of low* intensity are usually considered functional

The problem is emphasized by the fact that in 100 per cent of children one can record (1033) at least some slight systolic murmur Furthermore with sufficiently sensitive pick ups one can record a systolic murmur in all normal 20 year old subjects Finally, intracardiac phonocardiography and direct phonocardiography reveal a systolic murmur in or over the pulmonary artery in all cases With stethoscopy alone, the incidence of a systolic murmur is very high in certain age groups (Fig 224)

In the following discussion "functional" and "innocent" will be considered synonymous What murmurs can be *identified* as *'functional'* with fair certainty?

1 Circumscribed systolic murmurs such as (a) the late systolic variety (see Figs 225 to 229), especially if introduced by a systolic click (558), and (b) the mid systolic murmur which is separated from *both* the first and the second sound by a brief gap (Fig 230 and 231 also Fig 205) are quite clearly of extracardiac origin, probably from roughening of the pericardium, and are therefore innocent The circumscribed mid systolic murmur may have a grating quality (558) consistent with its origin in pericardial roughening It sometimes persists for several weeks or months or possibly indefinitely after acute pericarditis (Occasionally the murmur of aortic stenosis may show a gap between the first sound and its onset, and of course the usual gap after the murmur Possibly the presence of bundle branch blocks increases the likelihood of a gap preceding the murmur of aortic stenosis)

The late systolic murmur introduced by a systolic click illustrated by Brigden and Leatham (177) is probably an example of a pericardial murmur rather than of mitral regurgitation The fact that both may occur after rheumatic fever may cause confusion

The late systolic murmur has long plagued clinicians (596) and cannot honestly be said to be completely understood at present (704) In 1903 Hall (625) mentioned the view that it is due to mitral regurgitation with the valve competent only in the first part of systole an unlikely view Evans (441) emphasized its innocence The murmur *under discussion occurs* at the apex or left midprecordium If phonocardiograms show that

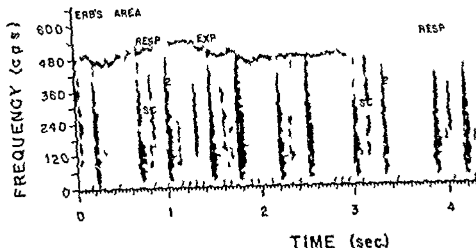


Fig 276 Innocent murmur and systolic click

D C (503601) 23 year old female was sent for examination because of an incidentally discovered *murmur* The murmur was present at the left sternal border only with the patient in the recumbent position *There was no past* history that seemed pertinent The character of the murmur (apex not shown)—mid systolic timing introduced by a click—is unmistakable evidence of its benignity A recording from Erb's area showed only the click

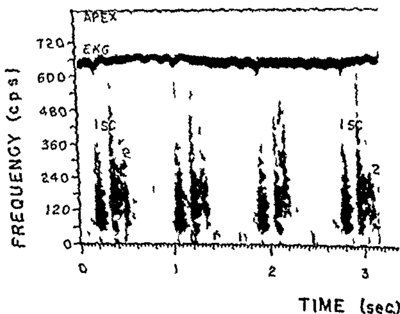


Fig 277 Extracardiac murmur and clicks

L H (731648) *34 year old male* was rejected for armed service 13 years before because of a murmur He had always been *active in athletics* and in his work as a farmer Detailed investigation including right heart catheterization revealed no *abnormality* Apex in left lateral decubitus The first of the systolic clicks now introduces a short murmur When a recording was made at the apex in the supine position there were multiple late systolic clicks

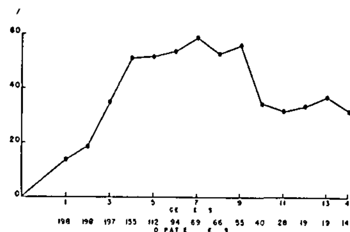


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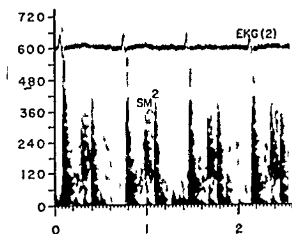


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The problem is emphasized by the fact that in 100 per cent of children one can record (1033) at least some slight systolic murmur Furthermore with sufficiently sensitive pick ups one can record a systolic murmur in all normal 20 year old subjects Finally, intracardiac phonocardiography and direct phonocardiography reveal a systolic murmur in or over the pulmonary artery in all cases With stethoscopy alone, the incidence of a systolic murmur is very high in certain age groups (Fig 224)

In the following discussion "functional" and "innocent" will be considered synonymous What murmurs can be identified as "functional" with fair certainty?

1 Circumscribed systolic murmurs such as (a) the late systolic variety (see Figs 225 to 229), especially if introduced by a systolic click (598), and (b) the mid systolic murmur which is separated from *both* the first and the second sound by a brief gap (Fig 230 and 231 also Fig 205) are quite clearly of extracardiac origin, probably from roughening of the pericardium and are therefore innocent The circumscribed mid systolic murmur may have a grating quality (598) consistent with its origin in pericardial roughening It sometimes persists for several weeks or months or possibly indefinitely after acute pericarditis (Occasionally the murmur of aortic stenosis may show a gap between the first sound and its onset and of course, the usual gap after the murmur Possibly the presence of bundle branch blocks increases the likelihood of a gap preceding the murmur of aortic stenosis)

The late systolic murmur introduced by a systolic click illustrated by Brigden and Leatham (177) is probably an example of a pericardial murmur rather than of mitral regurgitation The fact that both may occur after rheumatic fever may cause confusion

The late systolic murmur has long plagued clinicians (596) and cannot honestly be said to be completely understood at present (701) In 1903 Hall (625) mentioned the view that it is due to mitral regurgitation with the valve competent only in the first part of systole, an unlikely view Evans (441) emphasized its innocence The murmur under discussion occurs at the apex or left midprecordium If phonocardiograms show that

early systole is indeed free of murmur then the late systolic murmur can probably be considered innocent

Cotex (713) quotes his master Vaquez in the opinion that apical systolic murmurs are organic if holosystolic, non organic if mesosystolic. (Vaquez did think that ventricular dilatation can result in a protosystolic or telesystolic murmur.) Cotex emphasizes the fact that circumscribed systolic murmurs occur frequently following myocardial infarction. In fact he goes far to propose that such murmurs in mid or late systole occur with basal infarctions but when in early systole indicate apical infarction!

2 Murmurs with striking respiratory variation in a subject with a chest deformity which makes likely compression of lung tissue by the heart are probably cardiopulmonary (see Figs 206 and 207) and therefore innocent. Master and Stone (1049) found a systolic murmur in essentially all cases of

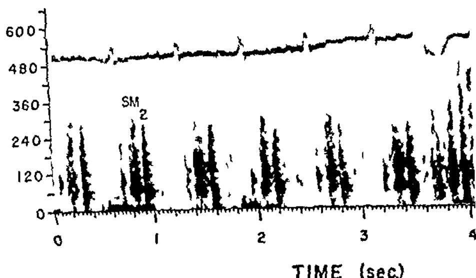


Fig 230 Extracardiac murmur
Circumscribed mid systolic murmur at the apex after acute pericarditis as part of hydralazine lupus (1534) in M McD (415a16). After persisting several months it disappeared completely. It had a rather croaking quality which is suggested by the tendency to harmonic organization.

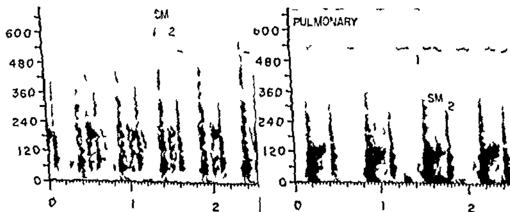


Fig 231 Fig 232

Fig 231 An extracardiac origin of this musical murmur in an 18 year old female patient with a history of possible rheumatic fever is suggested by the fact that it does not begin immediately with S_1 and probably runs over S_2 to some extent (from the sound S_2).

Fig 232 Musical systolic murmur in 18 year old patient with history of acute rheumatic fever. The musical murmur has the spectrographic pattern of the Still murmur.

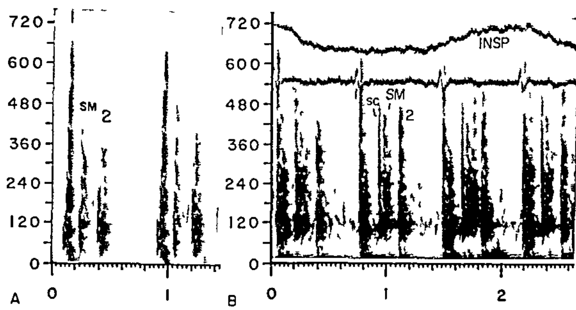


Fig 228 Midsystolic murmur introduced by systolic click

A Apex in patient (W B 674663) with Laennec s cirrhosis No abnormality of heart and surrounding structures at autopsy B LLSB in patient (J B 149692) with chest deformity (mainly kyphosis due to osteogenesis imperfecta)

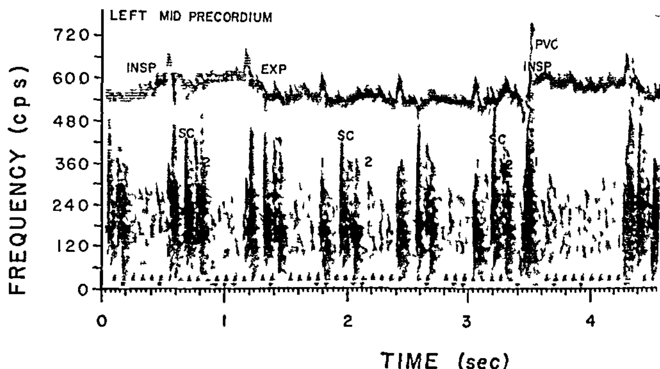


Fig 229 Syndrome of precordial pain palpitation cardiac neurosis and extracardiac sounds

This recording was made from the left midprecordium of W P (768996) a 38 year old white man who had had palpitations with a sensation of skipped beats for 10 to 15 years The palpitations always occurred when he was at rest but during periods of excessive fatigue or anxiety During the same period (and under the same circumstances) he had vague evanescent pains in the region of the left anterior axillary line Fifteen years previously he had been rejected for armed service presumably because of his heart but was given no further details His physician became much concerned about his condition when he heard a murmur which was however variable and placed him on digitalis As seen in the recording the murmur is circumscribed and introduced by a click—features characteristic of extracardiac origin One extrasystole is present (There is considerable continuous background noise either ambient or electronic between 120 and 240 c p s) The T wave changes present in the electrocardiogram may be caused by digitalis or represent residua of pericarditis

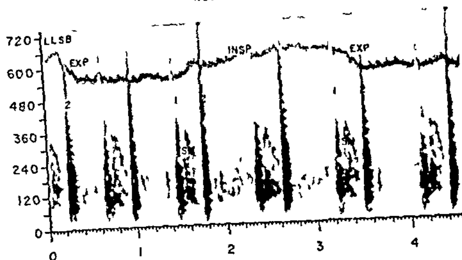


Fig. 234A. Functional systolic murmur.
LLSB in C. K. (72819), 16 year old male who is perfectly well but has a systolic murmur first discovered at the age of 8 years. The fact that the murmur is separated from *both* S_1 and S_2 may be significant as indicating its benign nature. At LLSB it is partially musical, a feature characteristic of the Still murmur.

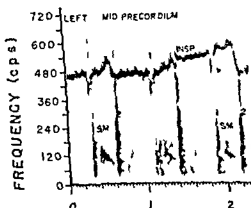


Fig. 234B. Functional murmur.
Left midprecordium in 7 year old boy (J. L. B34 at) with apparently normal circulation.

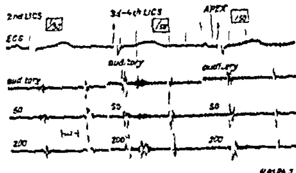


Fig. 234C. For comparison, oscillographic display of functional murmur Still type. The murmur is maximal at LLSB, is separated from both S_1 and S_2, and is musical as indicated by the regularity of its vibration. The three frequency channels are: ear-like, and two other with maximum response at about 50 and 200 cps respectively. (From Paulin and Mannheimer (1190).)

(C0) found with filtered recording that functional murmurs often had only relatively high frequency components whereas organic murmurs have both high and low components. Evan (439) had predicted as follows: "A detection of the frequency of the separate murmur may also contribute to a readier recognition of the innocent ones from amongst the others." This is essentially what the spectrogram does (see Figs. 232 to 236) and much less satisfactorily the oscillogram. Well (1520) who designated this murmur as the coarse variety of precordial murmur noted the regular musical characteristic of the vibration in the oscillogram and pointed out that the murmur may be very loud leading to a misdiagnosis of ventricular septal defect. Lau ng (1460) wrote that a child with a normal heart may have an extraordinarily loud functional murmur which is transmitted to the back. Logel (467) stated that Still's twanging string murmur is the most frequent variety of functional murmur, is rarely as intense as grade IV, occurs most often in the years two to four. Paulin and Mannheimer (1190) found that 43 per cent of functional murmurs were of this type.

funnel chest The murmur in some cases of chest deformity may be comparable to that produced in animals by compressing the pulmonary artery Often the murmur in milder cases of funnel chest can be eliminated by deeply held inspiration Presented in Figure 469 is a recording of a loud pulmonary systolic murmur produced by compression of the pulmonary artery by mediastinal lymphosarcoma the murmur could be eliminated by deep inspiration This is another instance of a functional murmur which was not innocent since it was caused by a grave condition

3 A venous hum (p 226), although loudest in the neck, may be audible over the upper precordium The fact that it is loudest in the neck and can be traced continuously from the neck to the precordium aids in its identification

4 Carotid bruit (p 227) may be heard at the base of the neck on the right in children and may suggest mild aortic stenosis The absence of the murmur in the aortic area assists in the correct identification

5 Mammary souffle (p 233) in pregnancy or the parturient state is easily differentiated if the clinician is familiar with the phenomenon

6 Still (1446) described, and others notably and most recently Harris and his collaborators (640 641 and 642) have studied the variety of systolic murmur which appears to be innocent and which is named for its resemblance to the twanging of a tight string The "twanging string murmur"[8] is indeed musical by spectrographic analysis suggesting that some elastic structure is thrown into vibration in systole Trigonoidation of the pulmonary cusps in systole, an entirely reasonable basis for a systolic murmur (262), results in three relatively taut flaps the pulmonary cusps It may be these that are incited to vibration A pericardial origin is also possible Ortiz (1167) has demonstrated the sound producing properties of normal rubbing serosae and drawn an analogy to the group of stringed musical instruments "Milk spots" roughened and thickened patches on the visceral pericardium have been suggested as sources of murmurs as far back as Gerhardt (541) in 1871 The musical character of this murmur is consistent with either trigonoidation or pericardial rubbing The murmur is referred to as "vibratory" by Harris (642), who points out its buzzing quality 'Groaning' is the adjective, perhaps even more appropriate, suggested by Lynxwiler and Donahoe (998) and found by them to apply to 500 of 620 basilar functional systolic murmurs and 120 of 620 apical functional systolic murmurs Stuckey (1451) stated that 40 of 145 innocent murmurs were squeaky or musical Among 300 children with systolic murmurs judged to be functional, Messeloff (1096) found that the murmur was musical in 55 per cent and in a further 6 per cent was blowing and musical The type of murmur under discussion, although possibly not representing a homogeneous group is probably the most frequent variety of functional murmur

Harris and his co workers (642) and Paulin and Mannheimer (1190) published oscillograms which suggest that Still's murmur is musical—its vibrations are regularly spaced—and does not begin quite with S_1 in many instances More recently Harris' group (643) has further confirmed the musicality by a spectrographic study Dunn (381, 387) arrived at similar conclusions from approximate estimates of frequency that there is a large group of functional murmurs characterized by restricted frequency span i e tendency to musicality and a tendency not to begin immediately with S_1 or extend completely to S_2 Bass *et al*

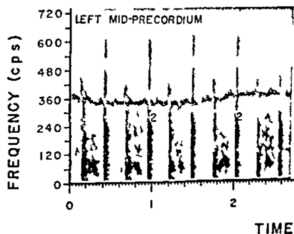


Fig 233 Groaning functional murmur
I M (B27352) 8 year old white male had a grade II mid systolic groaning murmur best heard at the left sternal border and varying with exercise and position The recording (from the left midprecordium) shows the characteristic musical quality

[8] Wedum and Rhodes (1518) called this the "fiddle string" murmur

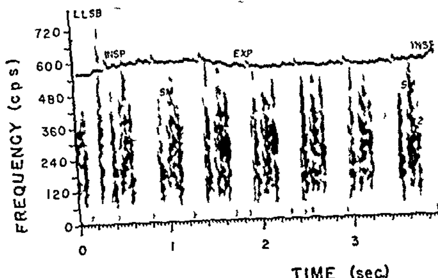


Fig 237 Semi musical extracardiac murmur
R P (73 428) 59 year old male was seen for manifestations apparently unrelated to the heart There was mild pectus excavatum and at the left sternal border there was a harsh somewhat musical systolic murmur loudest in inspiration and in the sitting position *SI CG* (LLSB sitting) Mitral and tricuspid closure sound seen with unusual clarity Gap before beginning of murmur musical element to murmur

7 The musical late systolic murmur which varies with respiration and may be introduced by a systolic click (p 207) is extracardiac in origin and benign in prognostic significance The case illustrated in Figure 237 is one such although the murmur is only partially musical

In young people with no anemia and with no evidence of dilatation of the pulmonary artery a murmur may be audible at the second third and fourth interspace at the left sternal margin The murmur is usually confined to the very first part of systole when the velocity of systolic ejection is maximal It is exaggerated by exercise and by the administration of adrenaline and amyl nitrate Rapid flow through the outflow tract of the right ventricle which at this site is relatively superficial is probably the main mechanism but trigonoidation of the pulmonary cusps is in addition an attractive possibility as a contributing factor Usually the murmur is loudest in the supine position (1096) and after exercise (889 1190) conditions which increase venous return and the stroke volume of the right ventricle The effect of both exercise and position is by no means distinctive since the murmur of valvular lesions may show the same change At least a faint murmur of this type in early systole when flow is most rapid can be recorded at the left sternal margin in the majority of young people especially after exercise and in the recumbent position and of course especially with a sensitive microphone (610) It is of note that in all subjects such a murmur can be recorded from the pulmonary artery by intracardiac phonocardiography or by direct phonocardiography (see p 84) The murmur heard in many instances is merely this normal murmur in somewhat exaggerated form As Hayer (1169) said ascribing the idea to Broadbent It is not the frequency of soft systolic murmurs at the pulmonary orifice that is remarkable What is remarkable is that they are not always present The fact that a murmur cannot be recorded directly from the inside or the outside of the aorta speaks against the view that innocent systolic murmurs of aortic origin are at least as common as innocent pulmonary systolic murmur in normal healthy children (1451)

Difficult to differentiate from the type of murmur which was discussed in the last paragraph and which probably can most legitimately be termed functional are the following (1) small atrial septal defect (2) bicuspid aortic valve

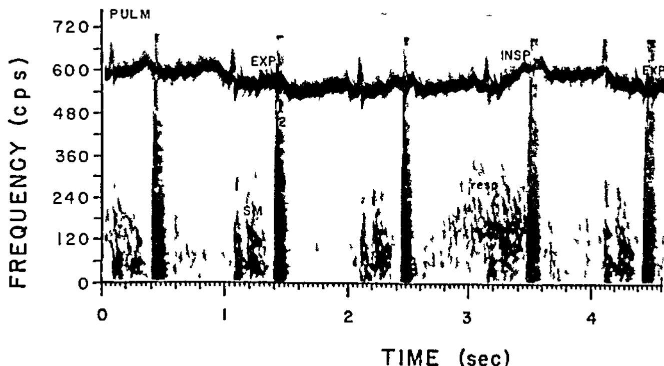


Fig 235 Functional systolic murmur

Pulmonary area in P M (643244) 16 year old female who had had nosebleeds and aching of the legs from the age of $5\frac{1}{2}$ years Because of a murmur the patient was referred with the diagnosis of rheumatic heart disease A systolic murmur at the left sternal border is rather groaning in quality The systolic murmur with a suggestion of musicality is separated slightly from both S_1 and S

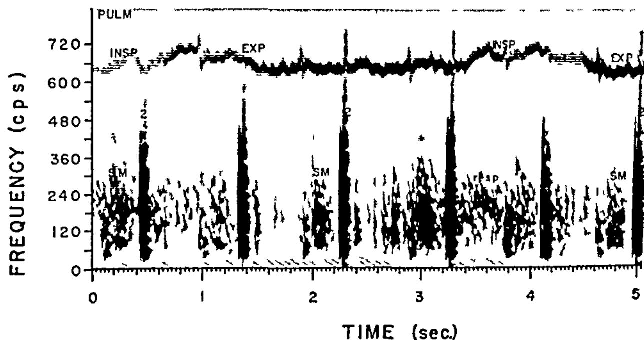


Fig 236 Functional systolic murmur

Pulmonary area in J M (353828) *17 year old female followed for five years for presumed* rheumatic heart disease the diagnosis being based almost exclusively on the presence of a systolic murmur of groaning quality The partially musical systolic murmur does not begin immediately with S_1 and is separated from S_2 by a brief gap A third heart sound is present

[illegible]	[illegible] clavicle	R[illegible]	Up [illegible]	Continu[illegible]	Cyclically variable	Machinery like	No special technique necessary	Pulmonary early systolic click [illegible]	Apical [illegible] murmur of relative mitral [illegible]
Functional murmurs ( ) Still	Left mid [illegible]		Up to [illegible]	Slightly separated from S [illegible]	Fundamental [illegible]	Groaning, creaking, whistling, twanging	Supine position	None	None
(d) [illegible] systolic	Apex		Up to 3		Wide band	May be rough	Some phases of respiration and some positions better than others	Systolic click may imitate	None

TABLE 11

Murmurs: a synthesis

	Main Area	Radiation	Characteristics: Intensity (Levine grade)	Characteristics: Timing	Characteristics: Pitch	Characteristics: Quality	How Best Heard	Associated Changes in Heart Sound	Associated Physical Signs
Mitral stenosis	Inside apex, well localized	Radial occasionally even to aortic	± to 4	Rumble may begin with OS; presystolic without atrial fibrillation	Low crescendo presystolic	Rumbling	Left lateral decubitus after exercise, bell lightly applied	M1 snapping, opening snap, P2 increased and pseudo split (*usually 12 + OS*)	Diastolic, especially presystolic thrill, Graham Steell murmur
Mitral regurgitation	Apex, wide area	Axilla, left back	1+ to 5	Holosystolic	Intermediate	Blowing, occasionally musical	No special technique necessary	M1 dull, protodiastolic gallop, P2 less regularly accentuated	If with stenosis, rumble louder than otherwise the case
Aortic stenosis	Noisy in aortic, musical in Irbs	Right base of neck to apex	1+ to 6	Christmas tree systolic	Wide band	Rough, rasping	Sitting leaning forward, full expiration	Dull or absent A2 (not invariable however)	Systolic thrill
Aortic regurgitation	*Irbs*	*Radial*	± *to 6*	*Decrescendo*, early diastolic	*High*	Blowing, purring, may be musical	Same as AS, diaphragm, naked ear sometimes best	A2 may be increased, normal or decreased	Occasional diastolic thrill, Austin Flint, aortic systolic murmur
Tricuspid stenosis	Left lower sternal border	*To left mid* precordium and apex	± *to 3*	As in mitral stenosis (see above)	As with mitral stenosis	Rumbling	Bell, recumbent inspiration	Tricuspid opening snap	Diastolic thrill, giant venous A waves, large RA, high VP, presystolic pulsations of large liver
Tricuspid regurgitation	Right lower sternal border	To right axilla and hepatic area	To 1 or	As in mitral regurgitation	Intermediate	Blowing	No special technique necessary	Protodiastolic gallop	Systolic pulse of liver veins

CHAPTER 11

Miscellaneous Varieties of Cardiovascular Sound

KOROTKOFF SOUNDS

The Korotkoff sounds (1241 1286) are heard over arteries of the extremities immediately below a compressing cuff which is inflated to a pressure above systolic pressure and then slowly deflated. The Korotkoff sounds are used in determining systemic arterial blood pressure. The level at which they first appear is taken as systolic peak pressure and the point of disappearance or marked change in tone as the diastolic level. The Korotkoff sounds at the upper and lower level are what we usually think of as "sound" that is they are transients. At intermediate level of cuff pressure the Korotkoff sounds are more murmurish. Korotkoff recognized three stages in the sound. Later five stages or tonal changes were described. The first phase is signalled by a tapping sound, the second by a softening of same, the third by a murmur, the fourth by a muffling of the sounds and finally the fifth by a total disappearance of sounds. It is the official recommendation of the American Heart Association (136) that the point of disappearance of sound be used as the most reliable index of diastolic pressure in most cases. It was stated that dulling of the sound (beginning of the fourth phase) is on the average 8 mm Hg above the true diastolic pressure. Systolic pressure as indicated by the first appearance of sound is 3–4 mm Hg too low on the average (136). The debate as to the most valid auscultatory index of diastolic pressure has raged from the earliest times (1019). In 1907 Ettinger (437) recommended disappearance; in 1909 Fischer (459) recommended muffling. Since the report of Bordley *et al* in 1951 (136) recommending disappearance, Roberts, Smiley and Manning (1285) have made a strong case for the use of muffling. They based their recommendation on a sizeable series of intra-arterial pressure recordings.

Recordings of Korotkoff sounds have been made by several workers (601). Several other workers (319 615 1569) have used the Korotkoff sounds as the basis for an automatic blood pressure recorder (427A).

In some cases of systemic arterial hypertension and/or generalized arteriosclerosis and sometimes in aortic stenosis there is so-called auscultatory gap (1190). The sound may be heard, let us say, between 240 and 200 and again between 150 and 125 with a silent period between 150 and 200 mm Hg. Occasionally a double gap, two silent periods, may be found (1295). Rodbard and Ciesielski (1294) found an auscultatory gap in cases of aortic stenosis and concluded that it corresponds to a plateau on the upstroke of the arterial pulse pressure wave. The gap could be widened by the application of a tourniquet to the forearm and eliminated by increasing blood flow with reactive hyperemia. In some cases of aortic stenosis there were double Korotkoff sounds; the occurrence of this phenomenon was related to the presence of a deep incisura on the upstroke of the arterial pulse pressure curve.

In aortic regurgitation and in states of marked peripheral dilatation as in anemia, thyrotoxicosis or fever, the Korotkoff sounds may be audible to zero, i.e. are audible without any compression of the artery. In none of these states is the diastolic pressure truly zero, although it may be only 30 or 40 mm Hg. What one is hearing is

Paulin and Mannheimer (1190) recorded a systolic murmur of at least faint intensity in 100 per cent of 108 children studied. In about half the cases intensity was sufficiently great that it was possible to classify the murmurs into two groups. (a) A "sinus-shaped" murmur separated from S_1 by a short gap (the Still murmur) was present in 43.5 per cent of the 108 children. (b) A protosystolic decrescendo murmur was present in 9.3 per cent. Both types were recorded at the left sternal border but those of type (a) tended to be lower whereas those of type (b) were in the second interspace.

Froment and Gonin (492) suggested that an abnormality in the development of the infundibulum, such as relative narrowing, might be responsible for a functional murmur in many instances. They colorfully referred to the infundibulum as the "veritable crossroads" of cardiovascular pathology. In a similar vein is the familiar reference to the pulmonary area as the "area of auscultatory romance," a designation which may have originated with Walshe (p. 23).

All seem agreed that the incidence of *murmurs in newborns* and in the first year of life (314) is very low but that the incidence increases steadily until in children and adolescents at least a slight murmur is demonstrable in the majority if not all. In a group of over 5000 newborns Richards (1270, 1271) found an incidence of systolic murmurs of only 1.7 per cent. The results of Lyons and his colleagues (999) were similar. The low incidence of systolic murmurs in the newborn increases the significance of any fetal murmur which may be heard or recorded before birth (see p. 255).

Murmurs in the newborn are in a few instances produced by congenital malformations; pulmonary stenosis and aortic stenosis are the particular deformities most likely to produce murmur from the beginning. Nadas (1137) states that of the systolic murmurs present at birth one in seven turn out to be on the basis of congenital heart disease. Even a grade III or IV murmur present at the left sternal border at birth may disappear completely in one or two months. Richards (1271) found that a murmur heard at birth carries a 1:12 probability of persisting as an indication of congenital malformation. When a murmur is first heard at one year of age the chance of its representing a congenital malformation is only 1:50. Taylor (1463) found an incidence of 4.8 per cent for systolic murmurs among the newborn. In the majority the murmur had disappeared by one year.

Boone and Levine (133) followed up a group of patients some of whom had murmurs labelled "organic," others with murmurs called "functional," depending on the basis of grade of intensity. It was found that cardiomegaly was ten times more frequent in the former type of case, partially justifying intensity as a criterion of significance. Kuttner and Markowitz (830) on following up a group of cases after eight years found that 48 per cent of those originally thought to have "organic" murmurs had evidences of heart disease, whereas the figure was 13 per cent for the group with "functional" murmurs. In Australia Stuckey, Dowd and Walsh (1452) concluded that of every ten school children with a murmur one has rheumatic heart disease, two congenital heart disease and seven functional murmurs.

The reports in the literature of a murmur interpreted as that of ventricular septal defect disappearing later probably were cases of functional murmur. For example F. Parkes Weber (1516) in an article entitled "Can the clinical manifestations of congenital heart disease disappear with the general growth and development of the patient?" described a child in whom a very loud systolic murmur with thrill was found at 14 months and at 2 years but there was absolutely no murmur at 5 years or at 10 years and the boy was passed for the Royal Navy.

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the pistol shot sound (see below) Usually there is to be heard a change in quality of the sounds when one goes from true Korotkoff to pistol shot sounds It is customary to record the pressure in such instances as 200/40-0 mm Hg for example, 40 mm being the level of change in tone Other peculiarities of the Korotkoff sounds (1) Giraud and Bert (554) described an apparent lowering of the diastolic pressure when the forearm was hyperextended (2) Finn and Geismar (921) found that in cases of aortic stenosis even when associated with aortic regurgitation the Korotkoff sounds disappear 30 or 40 mm Hg above the point at which oscillometric fluctuations are minimal This phenomenon is demonstrated by taking the blood pressure with an oscillometer rather than the usual device Its discoverers recommended it for the demonstration of organic aortic stenosis

Rodbard and his colleagues (1298) have recently shown by means of recordings that the lag period between events in the heart and the

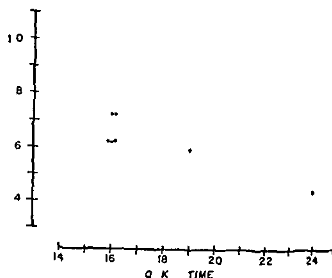


Fig 239 The relation between the interval from onset *of the QRS to the Korotkoff sound (abscissa in seconds)* and the duration of the previous cardiac cycle (ordinate in seconds) in patients with atrial fibrillation (Courtesy of Rodbard and Margolis (1296) and *Circulation*)

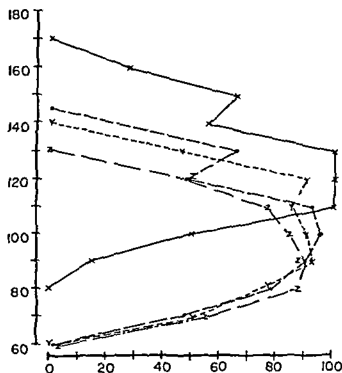
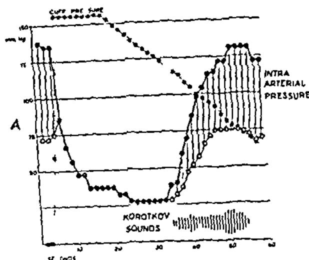


Fig 238 In four subjects with atrial fibrillation the percentage of heart cycles (as indicated by the electrocardiogram) which had representation in the form of a Korotkoff sound at the brachial artery are plotted on the abscissa for different cuff pressures *on the ordinate* (Courtesy of Rodbard and Margolis (1296) and *Circulation*)

onset of the Korotkoff sound varies with the pressure in the cuff As the pressure is lowered the QK interval, as they called the lag between the QRS and the Korotkoff sound is shortened Rodbard had shown earlier (1292) that the duration and intensity of the Korotkoff sounds afford an appraisal of the volume of blood flow to the extremity and that (1296) the time of onset of the Korotkoff sounds in the cardiac *cycle is related to the diastolic filling time* '

Ordinarily, in atrial fibrillation it is difficult even to estimate mean arterial pressure by the auscultatory method because of the variability in intensity of the Korotkoff sounds (Fig 238) Rodbard and Margolis (1296) indicate that in auricular fibrillation the determination of the incidence of the sounds [percentage of EKG cycles with sounds] at various cuff pressure levels permits a satisfactory measure of the blood pressure level despite irregularities of the pulse ' The QK interval in atrial fibrillation becomes shorter (Fig 239) and the Korotkoff sounds louder with longer diastolic periods probably because the velocity of the *pressure pulse wave* in the arterial tree is related to stroke output

The Korotkoff sounds are weakest when the blood pressure is determined with the arm dependent (93) Also slow inflation of the cuff and redetermination of blood pressure without com



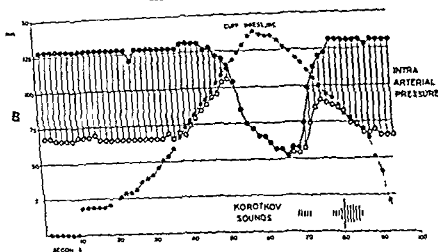


Fig. 240. Difference in Korotkoff sound with rapid and slow inflation of cuff. With slow inflation diastolic pressure rises in such a manner that no sounds are audible in an intermediate zone, i.e. an auscultatory gap is created. (From Ragan and Bordley (1239).)

plete deflation of the cuff in the interval result in weak Korotkoff sounds. Auscultatory gap (Fig. 240) is most likely to occur under these circumstances (1239). The common denominator is thought to be venous engorgement. These experiences further indicating that even though the level of blood pressure is not influenced, the intensity of the Korotkoff sounds is considerably dependent on the volume of blood flow to the arm. In an open tube the volume of flow is dependent on the difference in pressure at the two ends of the tube. The same principle obtains in the arm. Elevation of venous pressure reduces blood flow to the arm by reducing the pressure differential between artery and vein. The report by Rodbard and Margolis (1293) of auscultatory gap in arteriosclerotic heart disease is consistent with the other observations and with the conclusion that the volume of blood flow is an important factor in the intensity of the Korotkoff sounds. Reactive hyperemia eliminates the auscultatory gap. The gap can be induced in persons

not showing it spontaneously, by reducing total blood flow to the arm, for example, by occluding flow completely with a tourniquet of the forearm and determining the blood pressure in the conventional manner with the cuff on the upper arm. Amplification and recording of the Korotkoff sounds indicate that they are reduced greatly in intensity but do not disappear during the gap.

Galbraith (508) reported double Korotkoff sounds in one arm in a case of dissecting aneurysm of the aorta, presumably with double barrel configuration.

THE PISTOL SHOT OR WATER HAMMER SOUNDS

The pistol shot or water hammer sounds are heard over peripheral arteries in aortic regurgitation and occasionally in other conditions in which *peripheral vasodilatation is striking and/or* stroke volume is large, such as fever, anemia, pregnancy, and thyrotoxicosis. "Pistol shots" *can be produced in normal individuals by* administration of Apresoline or Priscoline or by inhalation of 10 per cent oxygen (839). The combination of increased cardiac output and peripheral vasodilatation is responsible. Generalized arteriosclerosis, especially aortic sclerosis, favors the appearance of the phenomenon. In all of these conditions, including generalized arteriosclerosis, there is a change in the shape of the arterial pressure curve which becomes steeper in both its anacrotic and catacrotic limbs with higher peak pressure and a generally narrow contour. The pistol shot sound is produced by the impact of this sharp pulse wave on the peripheral arteries. Quigstad and Steinert (1237) found a blood pressure of 200-210/50-60 mm Hg in a young woman with marked aortic calcification. "Pistol shots" tend to occur in such patients, despite the absence of aortic regurgitation.

As to mode of genesis, the "pistol shot" sound is probably fundamentally closely akin to the Korotkoff sounds discussed in the last section. The acoustical similarity is responsible for the fact that in aortic regurgitation and certain other conditions in which pistol shot sounds occur the diastolic blood pressure is usually given as "zero." The explanations which have been offered for these sounds are several (557): (1) sudden expansion of the vessel wall with resultant vibration and sound production,[1] (2) a "water hammer" effect due to sudden change in flow or pressure (433, 434, 814), (3) fluttering of the vessel wall due to Bernoulli effect (1292), (4) rapid change from one velocity profile to another (839).

THE SIGNS OF DUROZIEZ AND TRAUBE[2]

In aortic regurgitation and at times in the conditions which simulate aortic regurgitation as far as peripheral signs are concerned (fever, anemia, thyrotoxicosis, pregnancy, adrenaline administration), if the bell of the stethoscope is applied to a superficially located peripheral artery, such as the brachial, in such a manner that moderate compression is applied, one may hear a double murmur. The first of these occurs in normal subjects although it may be exaggerated in persons with the above mentioned disorders, and is the usual systolic murmur heard with partial occlusion of a peripheral artery, e.g., the Korotkoff sound. The second murmur is not usually present in the normal. The sign of Traube is a double *sound* (not a double murmur) heard over peripheral arteries in the same pathologic states as the Duroziez sign but *without* pressure of the stethoscope. It is much less frequently present than the Duroziez sign but the mechanism is probably closely akin.

It is the second component of the signs of Duroziez and Traube which presents the main problem in genesis. It is possible that it is produced by backflow past the site of narrowing in the artery. Backflow of some degree is demonstrated in the femoral artery, for example, by *McDonald and his collaborators* (668) under ordinary circumstances. Probably it is usually of insufficient degree to result in a murmur; under the conditions accompanied by the Duroziez sign it is presumably exaggerated.

On the other hand, Luisada (978) concluded that both sounds in the Duroziez and Traube signs are produced by forward flow. His evidence was derived mainly from two facts: (1) detail

[1] *Korotkoff himself subscribed to this view.*

[2] See reference 978.

compression of the artery exaggerated the phenomenon (2) both the first and second elements were transmitted down the vessel with approximately the velocity of the pulse wave Blumgart and Ernstene (123) thought backward flow was operative in aortic regurgitation but that in anemia fever and thyrotoxicosis and the situation when the arm is placed in warm water—states of peripheral vasodilatation—the sounds are produced with forward flow Blumgart and Ernstene (123) based these conclusions on the observations (1) that in aortic regurgitation the second element (diastolic murmur) was accentuated by pressure on the distal edge of the stethoscope bell placing the arm in cold water or applying a cuff inflated to a sub-diastolic level of pressure on the arm distal to the point of auscultation and (2) that in the non regurgitation group of conditions the diastolic murmur is increased by pressure on the proximal edge of the stethoscope and abolished by placing the arm in cold water or applying a cuff distal to the point of auscultation

Hale McDonald and Womersley (623) observed two velocity peaks in forward femoral arterial flow Assuming that these peaks are exaggerated in aortic regurgitation this observation may be evidence in favor of the mechanism proposed by Luisada On the other hand the observations of the same group on backflow in the femoral artery (663) might lend support to the mechanism advanced by Blumgart and Ernstene for the Duroziez sign of aortic regurgitation

FETAL HEART SOUNDS

The fetal heart sounds (397) consist of first and second sounds (Fig 241) as in the adult although one or the other may be quite faint Third and fourth sounds have not been identified to my knowledge Fetal heart sounds are first heard just above the symphysis pubis of the mother Later the area of the abdominal wall where the fetal heart sounds are most clearly audible is determined largely by the positioning and presentation of the fetus The rate is of course rapid (120-140 per minute) Fetal heart

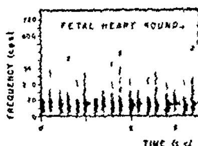


FIG 241 Fetal heart sound

sounds are usually audible at the fifth month rarely as early as the twelfth or fourteenth week

Prenatal diagnosis of congenital heart disease on the basis of a fetal heart murmur has been made by Simpson McCall and Kerr (1341) Padgett (1174) Dippel (3[illegible]) Roberts (1282) Berrey and Helman (92) and others (18) Ventricular septal defect is usually the lesion present (92 1462) Congenital heart block has been diagnosed in a few instances on the basis of the rate of the heart sounds (7 [illegible], 189) In one instance (1103) it was possible to establish that both twins were living by demonstrating two sets of fetal heart sounds at different rates

The number of workers who have applied themselves to the recording of the fetal heart sounds is large (3[illegible]0 6[illegible] 921 924 10[illegible]0 12[illegible]) *By* comparison the productivity has been slight Corner and Stran (291) have used the fetal heart sounds as the triggering mechanism of a ratemeter to use in studies of fetal distress A proper time constant (0 20 sec) insures that the meter is actuated only once per cardiac cycle

Croom (60[illegible]) demonstrated a systolic murmur in a recording of the fetal heart sounds and expressed the opinion that this would be entirely expected in all cases because of the patent foramen ovale and ductus arteriosus in the fetus It is doubtful however that these would generate sound atrial septal defects per se do not in the adult the ductus arteriosus is large in these cases and has essentially a normal relationship to the outflow of the right ventricle the physiologic polycythemia of the fetus tends to counteract murmur production In the fetal lamb studied with placental connections intact a faint systolic

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SECTION IV

Cardiovascular Sound in Disease

murmur produced at the patent ductus and transmitted down the aorta may be heard (137). The low incidence of murmurs in the newborn human (1270) increases the significance of any fetal murmur which is detected.

The funic, umbilical or fetal souffle, a murmur generated in the umbilical cord and having the rate of the fetal heart, occurs fairly frequently. At times it is only systolic, in other cases continuous. It is particularly likely to occur if there is twisting of the umbilical cord, for instance, around the neck of the fetus. The rate differentiates it from the uterine, or maternal souffle. Its changeable character aids in the difficult differentiation from a fetal cardiac murmur (355). Obviously the significance is quite different.

CHAPTER 15

Valvular Heart Disease

The classification of heart disease followed in this discussion is in essence that suggested by Hamman (629) and more recently by Harvey (640) for use in considering the causes of heart failure: valvular heart disease, myocardial disease, pericardial disease, systemic hypertension, pulmonary hypertension, congenital heart disease and miscellaneous disorders not properly placed in any one of the previous categories.

In the case of disease of each valve, stenosis and regurgitation may be the functional consequence of impairment of normal valvular functions, i.e., to open, allowing forward flow with a minimum of obstruction, and to close without back leak.

Rheumatic fever remains the leading villain in the causation of valve lesion. The order of frequency of involvement of the four heart

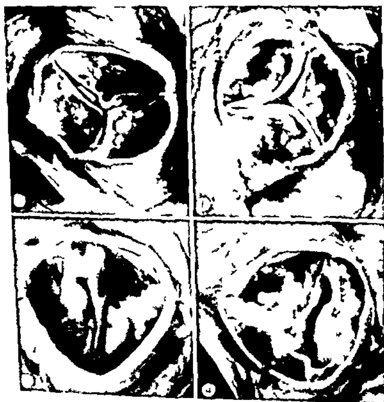


Fig. 247. Anatomic grades of calcific aortic stenosis.

Photographs of the valve from the aortic side. Grade I(a): moderate rigidity and calcification of the aortic cusps with minimal or absent commissural fusion. Grade II(b): moderate rigidity and calcification with fusion at one commissure. Grade III(c): marked rigidity but still with some flexibility, marked calcification, fusion of two commissures. Grade IV(d): pronounced calcification and rigidity without flexibility, fusion of at least two commissures, minimal residual ostium. (From Anderson, Kelsey and Edwards (21).)

valves is the same as the order of pre ures which the valves mu t sustain in the closed state (877 1479) From the lowe t incidence to the highe t the order of valve involvement i pulmonary (1 1 per cent) tricu pid (19 6 per cent) aortic (55 per cent) and mitral (86 per cent), the corre sponding pressures to which each of the valves is exposed at clo ure are approximately 5 24 72 and 116 mm Hg

AORTIC STENOSIS (AS)

ETIOLOGIC AND ANATOMIC CONSIDERATIONS (724) The long controversy between the two school one of which advocated athero clero is as the cause of calcific aortic stenosi and the other which advocated rheumatic fever (768) has been satisfactorily resolved by the concept (which has much to upport it) that rheumatic fever is one of the proce es which, by damaging the heart valve can prepare the soil for athero clerotic change and calcification (126) Congenital bi cuspid state of the valve is another (1404) and congenital aortic teno is which i now known to undergo calcification is a third Thi multi factorial or at lea t bifactorial ba is of calcific aortic teno is accounts well for the fact that i olated aortic steno is occurs predominantly in men in women mainly after the menopause in diabetic and in idiopathic hypercholesterolemia

Calcific aortic valve disease occurs unu ually often in as ociation with Paget s di ea e of bone (725B)

Various classifications of the grades of aortic steno is can be set up (21) The mildest grade i that variety in which the individual cu ps are rendered rigid by fibro is and calcification, but there i little or no fu ion at the commissures The severest grade i that in which complete fu ion has occurred with only a small opening in the center (Fig 242)

Both valvular and subvalvular (ubaortic) varieties of congenital aortic teno is occur as well as combined form Although the subaortic variety is of course ea ily identified at autop y and i clearly congenital the problem of dif ferentiating a congenital from a purely acquired basi can be difficult not only clinically but also pathologically e pecially in late stages when secondary calcifying atherosclerotic changes have taken place Description of a murmur from an early age—at least as early as four years by the criterion of ome—is taken as indication of congenital ba i Po t tenotic dilatation of the aorta occurs in all *types of aortic* steno i and occa ionally attain mammoth proportions (1081) Logue Bdinson and I (1081) described

Fig 243 Aortic tenosis with di ecting aneurysm of the a cen ling aorta

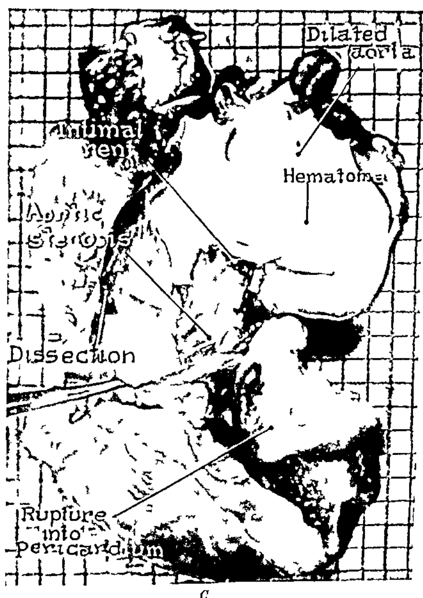
M I a white male student of theology age 2? had been known to have a heart murmur since the age of 3 There wa no hi tory of rheumatic fever and he ha l never ha l symptoms referable to the cardiova cular sy tem In 1948 an x ray of his chest showed dilatation of the a cen ling aorta with a heart of normal size (A *top left*) On the day of admi ion h ud lenly developed severe con tant dull aching substernal pain extending from the epigastrium to the neck The pain increa ed in everity an l he was a lmitted to the stu lent infirmary

Phy ical examination showed no evide ce of the Marfan syn lrome The blood pre ure was 106/84 mm Hg in both arm it was not recorded in the legs The heart was normal in ize The cardiac rhythm wa regular There was a harsh grade III sy tolic murmur accompanied by a sy tolic thrill at the first and second right intercostal pace The aortic econd ound wa replaced by a faint early high pitche l *blowing dia tolic murmur* The sys tolic murmur was transmitted over the precordium toward the apex The first sound at the apex was normal The y tolic murmur was not transmitted to the axilla No dia tolic murmurs coul l be heard at the apex The lungs were clear and resonant Liver and sp een were not tender or palpal le The remainder of the examination wa normal An electrocardiogram taken the day *after a lmi* ion showed left ventricular hypertrophy On the following day the electrocardiogram showed changes consi tent with acute pericarditis During his hospital stay the patient con tinued to complain of chest pain requiring opiates for relief During the following 3 days the pain became le s se vere 4 days after admi ion howe er there was an increase in severity of chest pain a ociated with su lden ga ping respirations that continued for 10 minutes when he died

Autopsy showe l aortic tenosis (B *top right and bottom*) with some calcification of the valve and with left ven tricular hypertrophy There was no evidence of rheumatic involvement of the mitral valve and the aortic stenosis was thought to be of cong nital origin There was a dissecting aneurysm which began with in the first few centi meters of the aortic valve in the area of dilated aorta There was retrograde exten ion of the di ection with *rup* ture into the pericardial sac Death occurred as a result of tamponade Microscopic examination showe l cystic medial necrosis of the aorta



A B



C

Fig 243 A, B and C

four cases of aortic stenosis and regurgitation in which cystic medial necrosis and dissecting aneurysm occurred in the ascending aorta (Figs. 243 and 244).

PHYSIOLOGIC CONSIDERATIONS. Were it not that pressure proximal to a stenotic aortic valve is increased, mean pressure and flow in the systemic circulation would not be maintained at a level compatible with life. Probably a systolic pressure differential of more than 150 mm. Hg between ventricle and aorta rarely exists, even in instances of most severe aortic stenosis. Gorlin and colleagues (578) found 150 mm. as the highest gradient in three patients whom they studied. The limiting factor seems to be ventricular systolic pressure, which seldom exceeds 250 mm. Hg. This ceiling is in turn imposed by the coronary flow necessary for the increased ventricular work. The interrelationship of pressure, flow and valve area is shown by these authors (578). A fixed stroke volume limit in a patient with pure AS in the absence of gross failure and often even in the absence of symptoms may indicate a tiny aortic orifice (578).

Valve orifice area (578) can be calculated*—provided there is *no appreciable* aortic regurgitation—by a *modification of the formula* used for a similar purpose in the case of mitral stenosis. Data of left heart catheterization are necessary.

$$AVA = \frac{CO/SEP}{44.5 \times \sqrt{LV_{SM} - BA_{SM}}}$$

where AVA = aortic valve area
CO = cardiac output
SEP = systolic ejection period in sec/min
LV_{SM} = systolic mean pressure in the left ventricle
BA_{SM} = systolic mean pressure in the brachial artery

Symptomatic pure aortic stenosis is usually found to have a valve orifice area of about 0.5 cm.

In all valvular stenoses a more meaningful index with reference to murmur production might be the valve resistance calculation by Poiseuille's law as used by Dow and colleagues (270). This calculation takes both pressure gradient and flow into account.

$$VR = \frac{(LV_{SM} - AO_{M}) \times 1332}{CO}$$

where VR = aortic valve resistance in dyne/sec/cm^{-5}
LV_{SM} = mean left ventricular pressure in mm. Hg
AO_{M} = mean aortic pressure in mm. Hg
CO = cardiac output in cc/sec. (This value must include regurgitant volume if there is any, i.e., it must represent total cardiac output.)

The shape of the aortic pressure curve is altered in a characteristic manner in aortic stenosis. The peak is delayed with the amount of delay being proportional to the severity of the aortic stenosis. Studies in models (244) indicate

* Gorlin formula is applicable only to small orifices, i.e., its validity cannot be tested on the normal valve orifice. There is a possibility that the constant in the formula derived empirically is in fact variable. The value of this constant is somewhat dependent on the form of the orifice (1303).

ysm was discovered as anticipated. After application of clamps and excision of the occluded portion of the aorta it was found that dissection had in fact occurred with a characteristic sheath-like double channel in the ascending aorta. The false channel was traversed by typical fibrous cords. The surgical procedure was performed according to plan without evidence of cardiac embarrassment. Soon after the clamp was removed from the aorta, pulsation of the heart became weak and ventricular fibrillation began. Cardiac massage and other efforts at resuscitation were of no avail.

At autopsy the heart weighed 900 gm., the excess weight being mainly the result of left ventricular hypertrophy. There was calcific aortic stenosis with fusion of one commissure and the valve also appeared regurgitant (Fig. 244 B). An aortic dissection extended from 4 cm. above the aortic valve through the end of the aortic arch and into the innominate and subclavian arteries. The thoracic aorta was of normal size. Microscopic study showed cystic medial necrosis in the ascending aorta and to much lesser extent in the subclavian, mesenteric and pulmonary arteries and abdominal aorta.



A



B

FIG. 214 Aortic stenosis with dissecting aneurysm of the ascending aorta

T. G. M. (726673), a 50 year old attorney, during his youth in a southern state had numerous episodes of acute tonsillitis but recalled no definite rheumatic fever.

At the age of 24 years he was first told that he had mitral stenosis. At the age of 35 years he was twice rejected for service in the Merchant Marine and in the Army because of murmurs. He was still asymptomatic. Exertional dyspnea had its onset at the age of 43 years and soon thereafter attacks of paroxysmal nocturnal dyspnea developed. Aortic aneurysm was discovered when he was 48 years old. Chest pain was never a conspicuous feature.

Physical examination revealed a blood pressure of 198/94 mm Hg in both arms, 235/108 in both legs. There was an active expansile systolic pulsation in a large area below the right clavicle and the same area was dull to percussion. In this same area, furthermore, there was a grade IV systolic murmur accompanied by a thrill and followed by a decrescendo diastolic murmur typical of aortic regurgitation. What seemed to be the same diastolic murmur was heard out toward the right axilla where it acquired a delicate high pitched quality, down the left sternal border and in the apical area where its quality was more low pitched and rumbling. The heart was strikingly enlarged to the left. Atrial fibrillation was present.

In general nothing about the patient's habitus or family history suggested the Marfan syndrome. The ophthalmologist could find no evidence of ectopia lentis.

Roentgenograms showed a large aneurysm of the ascending aorta and considerable enlargement of the left ventricle.

An operation similar to that used in the first patient was planned and at thoracotomy an enormous aortic aneu

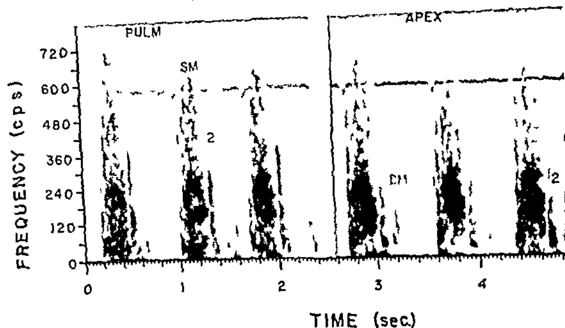


Fig. 21[illegible]. Full blown rheumatic aortic stenosis at the age of 7 years. S. S. ([illegible]) was first seen at $3\frac{1}{2}$ years with acute rheumatic fever manifested by pericarditis, changing murmurs, congestive failure. Smoldering of rheumatic fever continued thereafter. At the base (*left*) there are typical findings of aortic stenosis. At the apex (*right*) the systolic murmur is transmitted from the base and there is an early diastolic murmur which is probably of mitral origin since it begins after S_2 by a short gap.

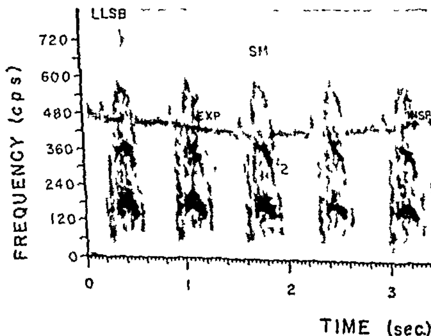


Fig. 218. LLSB in P. M. (*?30061*) with calcific aortic stenosis. Both noisy and musical elements are present with the latter predominant in this area. There is paradoxical *splitting* of the second sound which was better shown in the recording from the pulmonary area. The pulmonary closure sound is at the end of the murmur which stops slightly before the aortic closure sound.

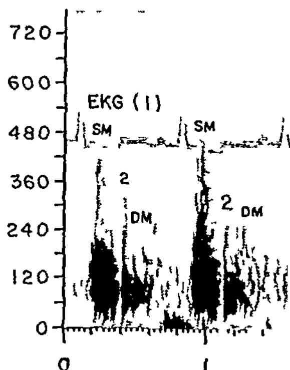


Fig 245 Aortic area in aortic *stenosis and regurgitation* on rheumatic basis Note Christmas tree configuration of systolic murmur gap between murmur and second sound (2) diastolic murmur decrescendo in both intensity and peak frequency

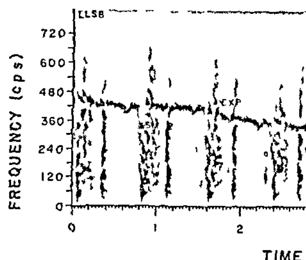


Fig 246 Mild aortic stenosis

C B R (231781) age 54 had palpitations at age 18 and was kept at bedrest for most of a year She had been plagued by tonsillar troubles most of her life The heart was at the upper limit of normal for size EKG showed left axis deviation and left ventricular hypertrophy A somewhat scratchy systolic murmur is present in the aortic area and transmitted into the base of the neck on the right The murmur stops well before S There is a presystolic gallop consistent with aortic stenosis The murmur has the appearance associated with scratchy sounds This is probably mild aortic stenosis

that the change in the arterial pulse pressure curve in aortic stenosis is not explicable on the basis of the stenotic orifice *per se* Instead, the basis must be sought in a change in the pattern of ventricular contraction

The end diastolic pressure in the left ventricle is elevated in cases of marked aortic stenosis, and *left heart catheterization usually reveals* giant *a* waves in the left atrial pressure pulse (562) *Hypertrophy of the left ventricle* undoubtedly results in change in the pressure volume *characteristics of the ventricle and* probably is responsible for the elevated end diastolic pressure in the absence of heart failure in the usual sense The giant *a* waves elevated end diastolic left ventricular pressure and the presystolic gallop of aortic stenosis are probably causally interrelated phenomena

In the case of both ventricles functional outflow obstruction caused by encroachment of hypertrophied myocardium on the outflow tract is being recognized with increased frequency By *left heart catheterization Brock (179A) finds* a gradient across the aortic valve area in cases of long standing systemic arterial hypertension This phenomenon represents a basis for an aortic systolic murmur in cases of hypertension Hancock *et al* (635) also write about "pseudostenosis" of the aortic valve in patients with a murmur typical of aortic stenosis associated with a thrill in two of five cases The basis for the murmur is not clear since left heart catheterization showed no "measurable systolic gradient across the aortic valve" *One certainly must* agree that there are cases with the murmur and thrill of aortic stenosis but no aortic obstruction *of physiologic significance* However the production of these physical signs in the absence of any gradient whatever across the aortic valve is puzzling Possibly in some cases fibrotic and rigid cusps without commissural adhesion vibrate vigorously in the outflow stream

CARDIOVASCULAR SOUND Usually the systolic murmur characteristic of aortic stenosis is *loudest* in the aortic area and is often accompanied by a systolic thrill although a thrill on the one hand is not essential to the diagnosis and on the other hand, may occur with relative aortic stenosis (see below) Occasionally especially in children

the diagnosis of aortic stenosis. Also the murmur is often well transmitted to the cardiac apex, thereby creating possible confusion with mitral regurgitation (Figs. 247 and 249). The latter is such a frequent occurrence that special emphasis is indicated (46, 522). At times the murmur is audible only at the apex (89). The shape of the murmur in the PCG (see p. 194) and the fact that it is nonholosystolic—being separated from S by a brief silent interval—permits differentiation from the murmur of mitral regurgitation.

Occasionally, especially with rupture of the posterior chordae tendineae of the mitral valve, the murmur of mitral regurgitation may be loud in the aortic area and even over the carotid arteries, resulting in simulation of aortic stenosis (see p. 000). There may even be a thrill in the aortic area because of the peculiar direction of the regurgitant jet from the mitral valve.

Occasionally there is an interval between the first heart sound and the onset of the systolic murmur (e.g. Fig. 249). Possibly this is more likely to be the case when left bundle branch block is present.

Usually the *quality* of the murmur of aortic stenosis can be described as harsh. Frequently it is musical (Figs. 248 and 249). When this is the case, three patterns may be seen. There may be a poorly transmitted musical murmur in the aortic area. In older diabetic patients the appearance and evolution of a musical systolic murmur of this type may be observed on *repeated* examinations over the years. A second pattern, which may be merely a later stage of the first, is the presence of a very loud musical systolic murmur heard over the entire chest and transmitted into the neck and even the extremities. A third pattern is diagrammed in Figure 176. In these patients one hears in the aortic area and at the base of the neck on the right a *conventional* noisy murmur. But at the left sternal border, over the left midprecordium and at the apex the *murmur* is purely musical. This dissociation is conveniently referred to as the Gallavardin phenomenon. The explanation offered for this situation is as follows: the noisy murmur probably represents the jet, the musical murmur the regular vibrations of *the* stenotic valve diaphragm. Since the aortic valve *underlies* the left sternal border and since the tense contracting hypertrophied left ventricle makes more intimate contact with the chest wall during systole—when the murmur is occurring—the radiation of the musical murmur is not surprising.

In 1897 Dickinson (343) wrote on the occurrence of musical mitral murmurs in connection with aortic stenosis. *One of his cases may have been an instance of extracardiac musical murmur on the basis of massive* cardiomegaly and/or pericardial affection. Another of his cases may have represented unusual transmission of the aortic murmur to the apex. However, he suggested another mechanism for a musical mitral murmur with aortic stenosis. In one case the aortic stenosis was so severe that peripheral pulses were not palpable and at autopsy water would flow through the orifice only in drops or small trickles. The mitral valve was normal. Dickinson suggested that the marked elevation of left ventricular pressure which was undoubtedly present during systole forced the normal mitral valve with production of a very small leak and a very high pitched murmur which he compared to the squeak of a mouse or guinea pig. He was aware, *however, of musical* murmurs produced by other mechanisms. "There may be musical murmurs of many kinds *as there are many* sorts of musical instruments." It would be difficult to prove the Dickinson mechanism for a musical apical systolic murmur in aortic stenosis; transmission of a *musical form of the* primary murmur seems most likely in such cases including Dickinson's.

There are some cases of calcific *disease of the* aortic valve in which very intense and highly musical systolic murmur is *heard* everywhere (the second type mentioned above) with none of the dissociation between the noisy murmur and the musical murmur—in fact with little noisy element. In these cases the evidence of aortic obstruction in terms *of the pulse* pressure and symptoms is usually minimal or at the most moderate. In such cases it is likely that there is fibrosis and calcification of the valve cusps with little adhesion at the commissures (Grade I or *II of Fig. 242*). There is therefore no jet, but a very intense murmur is produced through vibration of *the stiffened* cusps. Stokes (see p. 22) in 1855 was early to point out *this* paradox between murmur and clinical state in an old gentleman who complained "*that his* entire body was one humming top," although his general health continued *excellent*. In 1952 Schadt (1560) described an asymptomatic 58 year old farmer with calcific aortic stenosis and a systolic murmur audible 15 cm from the chest. The phys-

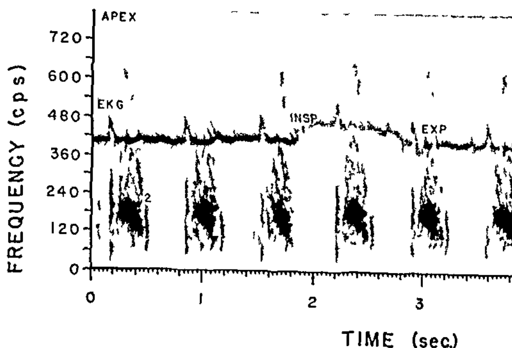


Fig. 249. Calcific aortic stenosis.

Apex in N. G. (320528), 74 year old male with gout. He had been thought to have mitral regurgitation (probably with calcified valve because the murmur is musical). The character of the murmur, especially the shape and frequency level of the harmonics and the gap before S_2 (which is probably aortic closure sound in this recording at the apex), are most consistent with calcific disease of the aortic valve.

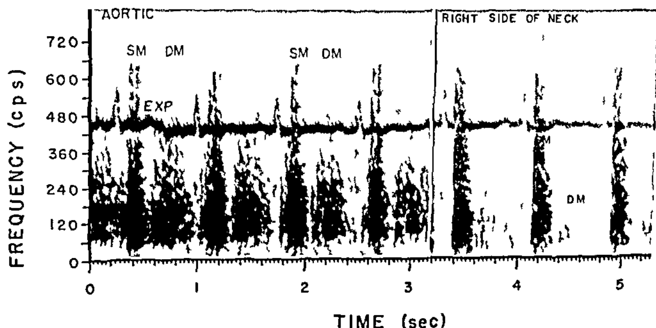


Fig. 250. Typical AS-AR with transmission to the neck.

In L. H. B. (761509), 44 years old, the findings in the aortic area (*left*) are completely characteristic. At the base of the neck on the right (*right*) the systolic murmur is late in onset, consistent with the view that the systolic murmur travels at the velocity of the pulse wave. The diastolic murmur is also faintly demonstrated.

and especially with the subaortic variety of aortic stenosis, the area of maximum audibility may be at the left costal margin. In such instances, ventricular septal defect may be a mistaken diagnosis. Customarily, the murmur is well transmitted into the base of the neck (Fig. 250) and to the suprasternal notch. In fact, in the absence of this feature there is reason to be suspicious of

cause of the fibrotic and calcific process which is likely to have occurred in the valve the second sound may be at least normally intense in spite of a reduced range of motion Although in general the aortic closure sound is reduced in very severe aortic stenosis this feature is not sufficiently consistent to be helpful in much less necessary to the diagnosis Occasionally the second sound displays paradoxical splitting (p 164) with the aortic component following the pulmonic and with the split greatest in expiration

Presystolic gallop occurs commonly with aortic stenosis as with other varieties of systolic overload of the ventricle Aortic regurgitation may of course be associated with aortic stenosis in fact more often than not at least some slight degree of aortic leak can be detected

See page 388 for a discussion of certain special features of congenital aortic stenosis

AORTIC REGURGITATION (AR)

(Syn Aortic incompetence insufficiency inadequacy etc)

ETIOLOGIC AND ANATOMIC CONSIDERATIONS (1372) The main cause of aortic valvular damage leading to predominant aortic regurgitation are rheumatism and syphilis *In addition* dissecting aneurysm Marie Strumpell spondylitis the Marfan syndrome idiopathic *cystic medial necrosis* of the aorta hypertension *atherosclerosis* senile ectasia of the aorta *congenital bicuspid aortic valve* other congenital malformation of the valve including ventricular septal defect with retroversion of one aortic cusp (see p 362) bacterial endocarditis and trauma are factors which alone or in combination may *lead to* regurgitation at the aortic valve

Severe predominant aortic regurgitation on a rheumatic basis *occurs more* commonly in men Bland and Wheeler (114) found this situation to be twice as common in men Review of cases of aortic *regurgitation* presenting for the Hufnagel operation turned up an unexpectedly large number of cases which appeared to be on a rheumatic basis Previously if asked how often rheumatic fever results *in* aortic regurgitation as an isolated or overwhelmingly predominant lesion most cardiologists would have expressed the opinion that this is rare

Castleman (246) described a case of marked fenestration of the aortic valve, with histologic evidence of cystic medial necrosis Matthews and Darville (1051) had a rather similar experience their patient probably had slight rheumatic involvement of the mitral and aortic valves and had had signs of aortic regurgitation before the development of an abrupt change in the diastolic murmur to one with a high pitched whirring quality audible without a stethoscope The patient died about 70 days after developing this murmur

The particular affection of the aortic valve known as retroverted cusp (Fig 252) and accompanied by a musical murmur is most often caused by syphilis In 1939 Bellet and colleagues (82) demonstrated that in syphilis loss of the fibroelastic basket on the ventricular surface of the aortic cusps permits retroversion (Fig 252C) Furthermore it is usually the right anterior coronary cusp which becomes retroverted

In the literature there is at least one autopsy proved instance of retroversion of an aortic cusp on the basis of cystic *medial necrosis of the* aorta (658) a second *probable case* is described (230) and I am following a patient who appears to represent a third (M B 701862) In the last patient a 57 year old man a very loud aortic diastolic murmur of strikingly musical quality was audible to both the patient and his wife No murmur of any variety was present on any of numerous previous examinations and the patient has remained essentially asymptomatic *during* more than three years of observation *since onset* of the musical murmur Serologic tests for syphilis including treponemal immobilization test are negative and the aorta is not *dilated* Matthews and Darville (1051) *described* spontaneous rupture with fenestration of the aortic valve apparently also on the basis of cystic *necrosis*

Exertion straining and blunt trauma to the chest may be the immediate precipitating factor in retroversion of an aortic cusp or in rupture in the sense of *fenestration* or separation of the attachment of a *cusp* A classical case is that described by Drew *and* Finlayson in 1879 Immediately after over exerting in connection with expulsion of an intruder *to his home* the patient noted a cooing in his chest It was audible also

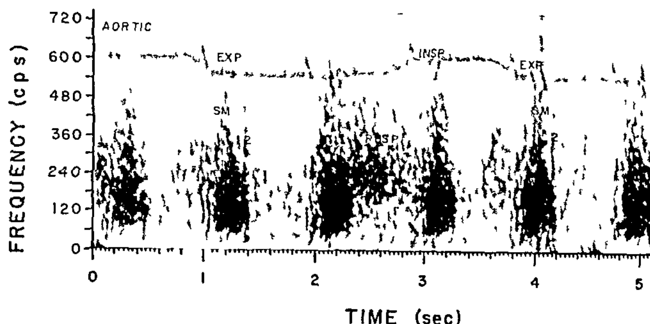


Fig. 251A. Effect of operation for aortic stenosis.
R. S. (734185), a 50 year old white male, had been having dyspnea, substernal tightness and syncope. On April 24, 1956 under hypothermia the aortic stenosis was surgically treated under direct vision. The main obstruction was produced by fusion of the two coronary cusps. One year after operation (see fig. 251B) the peak of the systolic murmur was much earlier. A minimal murmur of aortic regurgitation was demonstrated. (Much background noise in A.)

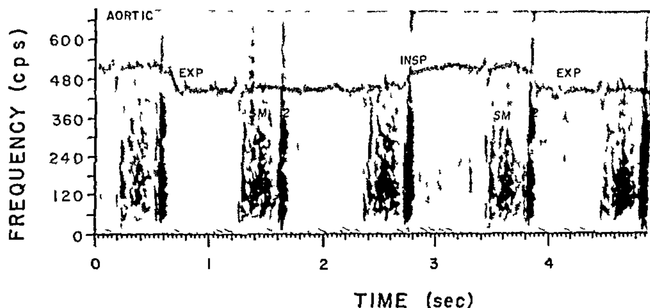


Fig. 251B

can first heard the murmur when examining the fully clad patient with the ophthalmoscope.

The *shape* of the murmur of aortic stenosis is characteristic. In the oscillogram it is diamond shaped because of the location of its intensity peak in mid systole; in the spectrogram it is shaped like a Christmas tree because of its frequency peak *in mid systole*. Furthermore the murmur of aortic stenosis stops before the aortic closure sound. There is usually a brief silent gap between *the end of the murmur* and the aortic component of the second sound. The shape of the murmur of aortic stenosis has adequate hemodynamic explanation (see Figure 98).

The *second sound in aortic stenosis* may be increased, normal or reduced in intensity. Be

to his wife. Autopsy revealed, in addition to retroversion of the right anterior coronary cusp of the aortic valve, typical intimal changes of syphilis in the ascending aorta, so syphilis was probably the underlying cause with trauma the immediate precipitating factor. These same physical factors operate in precipitating or exaggerating aortic regurgitation in the Marfan syndrome.

A convincing case of aortic valve rupture due to trauma was reported by Leonard, Harvey and Hufnagel (873) in a young man who was kicked in the chest by a stallion. In this patient rupture of the interventricular septum may also have occurred as suggested by the murmur and by the findings of two cardiac catheterizations. The possibility of rupture of a sinus of Valsalva into the right ventricle is an alternative possibility which would account for all findings. Usually when traumatic rupture of an aortic valve occurs there is pre-existing syphilis, rheumatic affection, bacterial endocarditis or the Marfan syndrome.

⁴ In 1879 the typical gross appearance of syphilitic aortitis was not generally recognized. MacCallum (1002) wrote as follows: "It was impressive and a revelation to ... in 1903 at the meeting of the German Pathological Society to the discussion by Chiari and others of the discovery by Doehle and Heller of the syphilitic nature of these changes and particularly to realize the perfectly characteristic and peculiar appearance of the aorta so affected. Since then everyone has recognized it at a glance. It seems to be one of those simple triumphs of observation that make one ashamed to have been so blind."

Proudfit and McCormack (1235) described an instance of aortic valve rupture in a hypertensive man who was in an automobile accident. Kissane, Koons and Clark (797) reported two convincing autopsied cases of traumatic rupture of the aortic valve. In 1928 C. P. Howard (711) was prompted to his classic review of traumatic aortic regurgitation by the case of a 33-year-old chauffeur who developed a thrilling in the chest following the strain of cranking a car. Two Wassermann reactions were negative.

Mazzitello (1044A) described a 17-year-old man who was kicked in the chest during a fight. Three months later a grade 3 diastolic murmur audible all over the chest was loudest at the apex. The patient died in heart failure nine months after the accident. At necropsy the aorta showed a T-shaped tear just above the posterior cusp of the aortic valve. A false passage extended from there to a point just proximal to the innominate ostium. I have observed a similar case in a 44-year-old man who developed aortic regurgitation after a load of bricks fell on his chest (J. M. 611,512).

I have seen one patient (M. G. 321967, aut. 22067) in whom a luetic aneurysm at the base of the aorta ruptured behind the posterior aortic cusp into the left ventricle producing signs of aortic regurgitation (258, Fig. 6).

Arteriosclerosis (or atherosclerosis) probably can rarely, if ever, be assigned as the primary cause of rupture of an aortic cusp productive of a musical diastolic murmur. One case reported as such in a series (228) of non-traumatic rupture of the aortic valve actually had syphilis. The pa-

Fig. 25. Retroverted right anterior coronary aortic cusp caused by syphilis. A. Artist's representation. Note the separation of the commissures, "tree barking" of the aortic intima, relatively high position of the coronary ostium. The right anterior coronary cusp is involved. B. Photograph of specimen showing same features. W. H. (J.H.H. 50600), a 50-year-old Negro male, was admitted to the hospital because of mild pulmonary edema. Syphilis and treatment for same were denied. Six weeks before admission the patient was first seen in the Emergency Department because of pulmonary edema. At that time a loud diastolic murmur of non-musical quality was present. Blood pressure was 160/40 mm Hg. When next seen and admitted to the hospital blood pressure was 130/30-0 mm Hg and the diastolic murmur was now the typical loud cooing or sea gull sound.

X-rays revealed pronounced enlargement of the left ventricle without evident aortic dilatation. Serologic tests for syphilis were positive and the spinal fluid complement fixation reaction for syphilis was also positive. Numerous blood cultures were sterile and the patient remained afebrile.

The patient demonstrated progressively more severe manifestations of congestive failure and died 11 months after appearance of the sea gull murmur. Autopsy revealed retroversion of the right anterior coronary cusp of the aortic valve and relatively little change in the other cusps. The sinus of Valsalva corresponding to this cusp was moderately dilated as were the other sinuses to a lesser extent. The commissures were slightly separated and the coronary ostia somewhat high but normally patent. Mild intimal changes typical of luetic aortitis were present throughout the thoracic aorta.

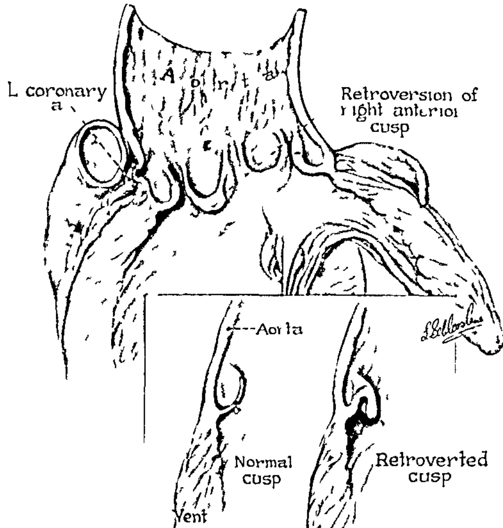


FIG 252 A

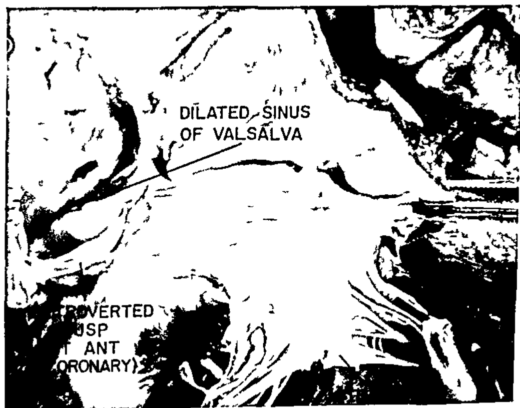


FIG 252 B

FIG 253 Fenestration of semilunar cusps

Aortic (above) and pulmonic (below) valves in 68 year old man with cor pulmonale A grade 2 diastolic murmur was heard at the left sternal border (Courtesy of Friedman and Hathaway (1814) and the American Journal of Medicine)

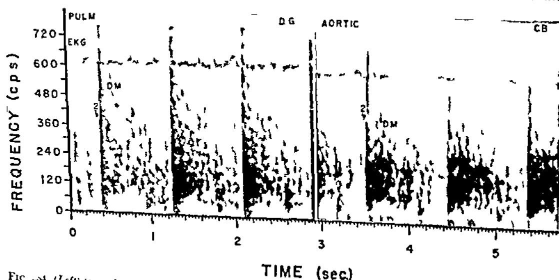


FIG 254 (*Left*) typical aortic regurgitation (rheumatic) Pulmonary area An aortic diastolic murmur was heard within three weeks of the onset of what was thought to be the first attack of ARF at age 10 in D C (405787) The patient was asymptomatic at age 21 and participated actively in sports as a basketball coach (*Right*) typical aortic regurgitation (rheumatic) complicated by SBE Aortic area in C B (532713)



Fig 25[illegible] C Retroverted aortic cusp
Destruction of the elastic fibers which sweep up onto the ventricular aspect of the valve and normally represent a retaining basket is demonstrated (From Bellet, Gouley, Nichol and McMillan (82))

tient, a Negro laborer, was seen at the Johns Hopkins Hospital in 1901 by Drs William Osler, Rufus Cole and Thomas McCrae There was musical aortic diastolic murmur which Dr McCrae compared to the bass string of a cello and which was audible six inches from the body The autopsy was performed by Dr William C MacCallum who rendered the final diagnosis of rupture of an aortic cusp due to arteriosclerosis (It was recorded In connection with the musical murmur it is of interest to note the finding of a cusp of the aortic valve which flapped back and forward and was probably the causative factor) However by the statement of MacCallum himself (see footnote p 271) syphilitic aortitis was not recognized in 1901 Furthermore recent review of the histologic sections reveals absolutely typical syphilitic aortitis

So called rupture of the aortic valve may occur in the course of bacterial endocarditis

Fenestrated semilunar valve cusps were found in 72% of 342 hearts by Friedman and Hathaway

Fig 253 Fenestration of semilunar cusp

Aortic (above) and pulmonic (below) valves in 68 year old man with cor pulmonale A grade 2 diastolic murmur was heard at the left sternal border (Courtesy of Friedman and Hathaway (48a) and The American Journal of Medicine)

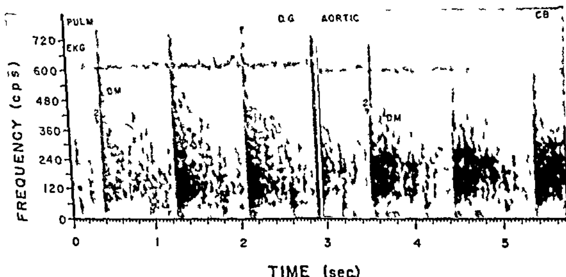


Fig 254 (*Left*) typical aortic regurgitation (rheumatic) Pulmonary area An aortic diastolic murmur was heard within three weeks of the onset of what was thought to be the first attack of ARF at age 10 in D C (40787) The patient was asymptomatic at age 21 and participated actively in sports as a basketball coach (*Right*) typical aortic regurgitation (rheumatic) complicated by SBE Aortic area in C B (532 43)

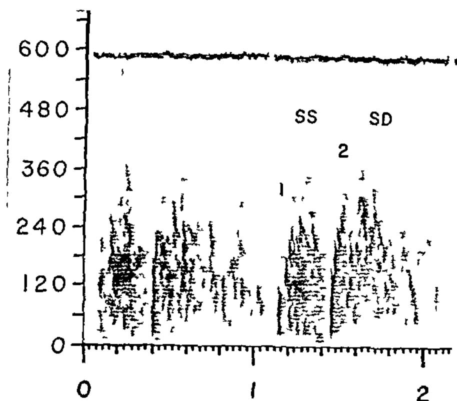


Fig 255 LLSB in case of typical *aortic stenosis and regurgitation* There is a gap between the end of the Christmas tree murmur (SS) and S2 The diastolic murmur (SD) is briefly crescendo then decrescendo

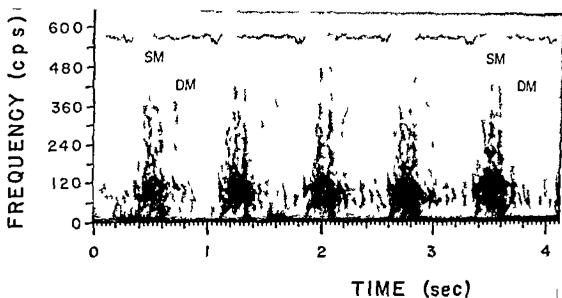


Fig 256 Demonstration of minimal aortic regurgitation in presence of aortic stenosis

Left sternal border in patient with rheumatic aortic stenosis and regurgitation A systolic murmur of typical shape is present An unusual feature is the continuation of the murmur to one component of S_2 which is probably pulmonary Left bundle branch block is present with paradoxical splitting of S_2 i e the pulmonary component precedes the aortic The associated aortic stenosis exaggerates the paradoxical splitting A second feature of note is the unusually faint high pitched nature of the aortic diastolic murmur The spectrogram can more easily and accurately demonstrate a faint diastolic murmur in the presence of a loud systolic murmur than can the oscillogram (see p 81)

(1844) They believe that a form of atrophy which may begin in early childhood and be exaggerated by aging dilatation of the ring and increased intravascular pressure is responsible Aortic regurgitation with systemic arterial hypertension and the Graham Steell murmur of pulmonary hypertension may frequently have fenestration of semilunar cusps as the anatomic substrate

The question of whether aortic regurgitation can occur on the basis of myocardial weakness during heart failure has been discussed on page 163

Acute dissecting aneurysm may cause aortic regurgitation through distortion of the aortic ring by the medial hematoma (p 452) but often the mechanism of the association between aortic regurgitation and dissecting aneurysm resides in the fact that the two have a common basis — idiopathic cystic medial necrosis the Marfan syndrome hypertension

In cases of aortic regurgitation *one is likely to* see diastolic pockets or *swallows nests directed* toward the aorta and located on the *interventricular* septum a short distance below *the aortic valve* Sometimes called in the older literature Zahn's (1601) or Schmincke's (1362) pockets, these represent a scuffing up of the endocardium by the regurgitant stream (Rarer are *pockets* facing downward in association with aortic stenosis and pockets in the left atrium facing the mitral valve in mitral regurgitation) Particularly in cases with an Austin Flint murmur impact of the regurgitant stream on the *aortic* leaflet of the mitral leaflet may *produce a jet* lesion there (581) A jet lesion may *develop on* the intima of the ascending aorta in *aortic regurgitation* merely because of the large stroke volume and sharp concentrated *forceful* ejection by the ventricle It is *important to recall* that syphilis does not cause *aortic stenosis* in the actual organic sense

PHYSIOLOGIC CONSIDERATIONS (1523A) Obviously the *regurgitation of blood into the left* ventricle results in an *increase in left ventricular* stroke volume since under *conditions of adequate* function something approaching a normal quota of blood plus that *volume regurgitated* during previous diastole is *ejected with* each ventricular *systole* The velocity of ejection is increased since there is a greater volume of *blood* to be ejected in roughly the same period of time and since the pressure into which the ventricle is facing is low at the beginning of ventricular ejection

The collapsing pulse and bobbing head of aortic regurgitation are principally a function of the shape of the arterial pulse pressure curve (1541) There is what Wiggers terms "systolic collapse" of the arterial pulse One wonders whether regurgitation at the aortic valve might not begin before the second sound—at least before the pulmonary component of the second sound The late systolic element of the musical murmur shown in Figure 97D might be evidence for this

Lowenberg (966) refers to cases in which an aortic diastolic murmur present during a *period* of heart failure disappeared when *compensation was re-established* Assuming that this was indeed aortic regurgitation and not pulmonary

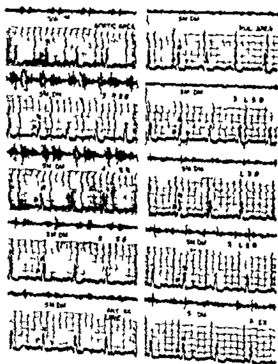


Fig. 2?? Ruptured aneurysm of sinus of Valsalva in a 48 year old woman. The murmurs of aortic regurgitation were best heard along the *right sternal border* (RSB) than along the left (LSB). SM systolic murmur DM diastolic murmur (*Courtesy of Harvey* (6??) and *Circulation*)

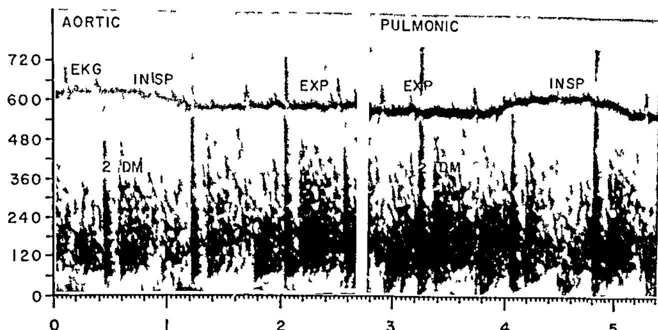


Fig 258 Aortic regurgitation with gap separating S_2 and onset of decrescendo murmur
Aortic and pulmonic areas in V F (413145) 21 year old female patient with rheumatic valvular heart disease predominantly aortic regurgitation and mitral regurgitation Although not entirely evident the gap between the second sound and the diastolic murmur is striking Timing with the electrocardiogram and general appearance make it clear that the sound marked 2 is indeed S_2 and not a late systolic click which could create a false impression of gap

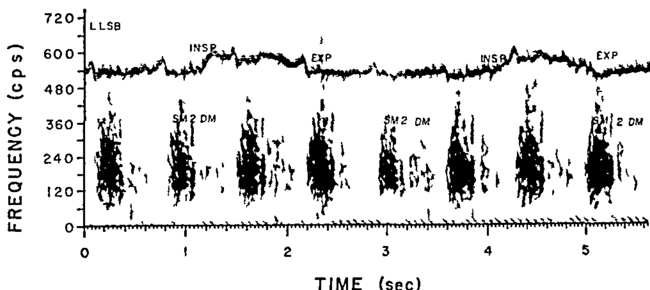


Fig 259 Gap between S and murmur of aortic regurgitation (LLSB) in C G (744355) who had left bundle branch block The combination of left bundle branch block and aortic stenosis causes paradoxical splitting of S_2 The Christmas tree murmur of aortic stenosis extends to the pulmonary closure sound The diastolic murmur begins only after a brief gap The aortic closure sound is poorly demonstrated

regurgitation, this functional aortic insufficiency" could be so identified by comparison of the blood pressures in the arms and legs Normally there is little discrepancy in the pressures nor is there much in cases of functional insufficiency even though the diastolic pressure is depressed In organic regurgitation systolic pressure was 50 to 100 mm Hg greater in the legs I suspect this differential is spurious The difference between the two types of regurgitation

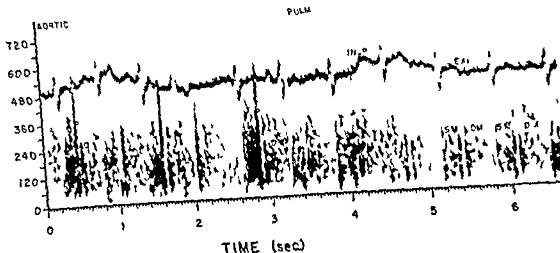


Fig. 260. Gap between S_2 and murmur of AI.

Aortic (A) and pulmonary (B) areas in J. S. (60830), 21-year-old female said to have had a murmur from birth. EKG showed marked left ventricular hypertrophy. The [illegible] paradoxical splitting, it decreases with inspiration.

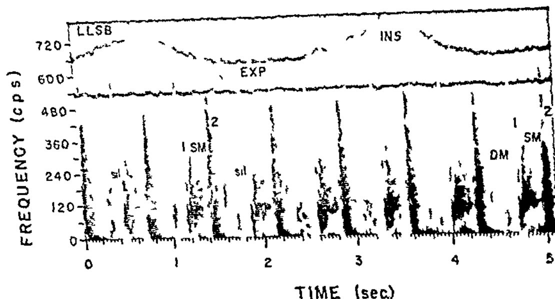


Fig. 261. Interruption of diastolic murmur by atrial systole.

A. C. (59906) [illegible] died at the age of 44 years with the anatomical diagnoses of calcific aortic and mitral stenosis. Regurgitation at these valves was thought to be present also. SPCG (LLSB): A systolic murmur is introduced by an early systolic click. Pulmonary closure is very loud and is preceded by a faint aortic closure sound. A murmur begins immediately with the second sound and ends with a transient which may be either atrial sound or closure sound. There was sinus rhythm and normal PR interval in this case because of [illegible] (p. [illegible]) the electrocardiogram does not show the P waves clearly and shows artifactual waves that suggest P waves. Probably atrial systole increases ventricular pressure to the point that there is no longer sufficient aorto-ventricular pressure gradient to maintain the murmur.

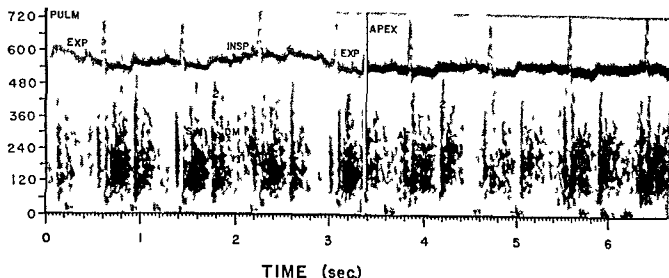


Fig 262 Presystolic gallop and Austin Flint murmur

Apex (A) and pulmonary area (B) in O B (751996) with syphilitic *aortic regurgitation* There is in addition to the early diastolic murmur a systolic murmur probably of relative aortic stenosis a striking atrial gallop and an Austin Flint murmur at the apex

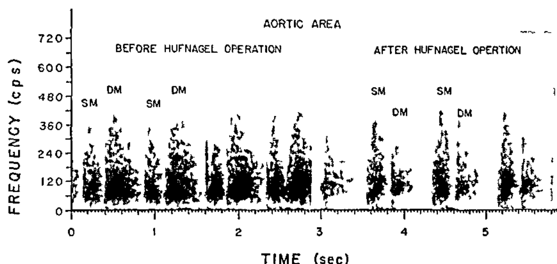


Fig 263 Effect of Hufnagel operation

The aortic diastolic murmur of aortic regurgitation (rheumatic) before and after Hufnagel operation in B C (474071) Intensity calibrated SPCG s recorded and analyzed in an identical fashion The systolic murmur is little changed

probably is caused entirely by *differences in* volume of regurgitation—or it may have been pulmonary regurgitation that was present in the group thought to have relative aortic insufficiency

Left heart catheterization in patients with aortic regurgitation (1116) and observations in animals with an experimentally established model of aortic regurgitation (1347) indicate that in some cases of very large regurgitation the end diastolic pressure in the left ventricle may exceed the pressure measured in the left atrium at any time in the cardiac cycle Obviously this observation has several important implications for cardiovascular sound (1) It is conceivable that the mitral valve will close before the end of diastole and before ventricular systole This could be a silent process if it occurred slowly as one would expect to be the case (2) In those cases in which the valve is already closed at the onset of ventricular systole the first heart sound would be expected to be muffled (3) Cases would be anticipated in which an intermediate grade of elevation of left ventricular end diastolic pres

ure exists and pressure in the left atrium with atrial systole may temporarily exceed that in the ventricle The result might be expected to be the movement of blood through a narrowed mitral orifice with the production of a presystolic murmur—? the Austin Flint murmur

Regurgitant flow rarely exceeds forward flow The combination of mitral regurgitation with aortic regurgitation results in a decrease in the effective ventricular output according to the studies of Sarnoff and colleagues (171A)

CARDIOVASCULAR SOUND It is proper to speak of the murmurs of aortic regurgitation, because as will be noted the systolic murmur is as much an integral expression of the pathologic physiology of this lesion as the diastolic murmur and the Austin Flint murmur occurs frequently However the characteristic and pathognomonic murmur is the arterial diastolic murmur previously described in detail (see p 197)

The location of maximum audibility of the pathognomonic murmur is usually Erb's point but occasionally may be down the right sternal border at the xiphoid, at the cardiac apex or even in the left axilla It is not yet clear how much significance in terms of etiology of the aortic regurgitation or precise anatomy of the valve lesion can be assigned to the radiation of the murmur Some have claimed that predominant radiation down the right sternal border occurs more commonly in syphilis (493 and 606) the Marfan syndrome coronary sinus aneurysm (Fig 256) and dissecting aneurysm of the aorta (1,00) than in rheumatic aortic regurgitation Also specific aortic valve lesions in bacterial endocarditis may result in a murmur down the right sternal border The practice of auscultating down the right sternal border should be cultivated Evan and Cuinard (926) speak of the rare cases in which an aortic diastolic murmur is heard only in the first right interspace It is worth while emphasizing that aortic diastolic murmurs of more than minimal intensity are transmitted to the apex rather as a rule Quite aside from the Austin Flint murmur there is opportunity for

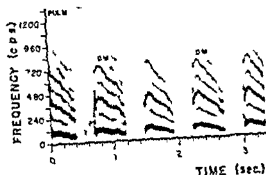


FIG 263A Reinverted aortic cusp from syphilitic in T S (103183) 50 year old male Of note are the following features (1) an unusually large number of harmonics are present (pulmonary area) (2) there is at the time of atrial systole an increase in the downward slope of the harmonics (cf pulmonary area) at the apex there is an Austin Flint murmur which is partly musical See Fig 263B

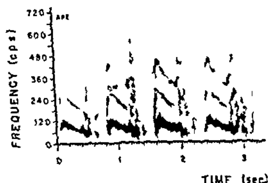


FIG 263B See Fig 263A

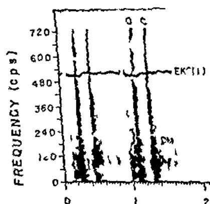


FIG 264 After Hufnagel operation

Recorded after Hufnagel operation in the same patient as in Figure 263 from area where the sound of the artificial valve were loud Note delay between beginning of QRS and valve opening sound (o) On the other hand the valve closure sound (c) almost coincides with S₂ Systole beyond the valve is abbreviated

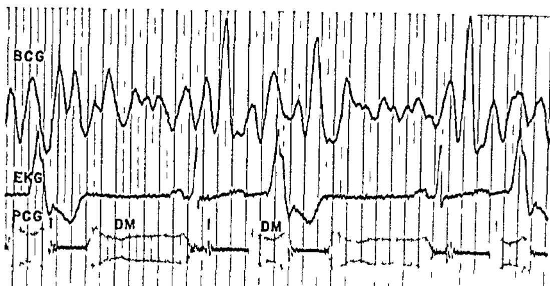


FIG 266 Retroverted aortic cusp

Oscillogram in patient M G (124569) Only the fundamental is demonstrated The diastolic murmur shows interesting changes in intensity At its onset it takes a brief time to attain maximum intensity and is decrescendo thereafter until mid diastole when there is a second increase in intensity ventricular filling probably pushes the retroverted cusp into the regurgitant stream A third rise in intensity occurs with atrial systole—probably for similar reasons—and is followed by a sharp decline in intensity probably because of abrupt decrease in the aorto ventricular pressure differential There is bigeminy caused by digitalis intoxication and with each extrasystole a sharp increase in intensity of the diastolic murmur occurs *before it is abolished again probably because of displacement* of the retroverted element into the regurgitant stream Waxing and waning of the musical aortic diastolic murmur was noted by Gelfand and Bellet (537 Fig 6) and attributed to the retroversion of two aortic cusps

Although autopsy examination is not available to complete the evidence this patient was studied with such care over a period of 20 years that the evidence for non luetic retroversion tear or perforation of an aortic cusp is convincing *As in this patient abrupt onset of awareness by the patient of a musical sound in the chest occurred in a* number of the patients with retroversion of an aortic cusp on the basis of syphilis

M G (J H H 124569) a Negro female was 54 years old when her sixth admission to the hospital was prompted by the development of a purring sound in her chest four days previously

She had been well most of her life and previous hospital admissions were for obstetrical deliveries Repeated serological tests for syphilis were always negative there were never previous manifestations interpretable as syphilitic in etiology and no therapy for syphilis had ever been given

In 1938 (at the age of 40 years) during her last pregnancy the blood pressure was 160/110 mm Hg and cardiomegaly with signs of mild congestive heart failure was present Tubal ligation was performed Two years before final admission chest x ray revealed slight enlargement to the left and there was left axis deviation by electrocardiogram The blood pressure on repeated examinations during this period was always normal or only slightly elevated No diastolic murmur was heard

One week before admission she was awakened with a sensation of fullness in the substernal area accompanied by nausea These symptoms passed off quickly and the patient felt well until four days before admission when while sitting and reading the paper after supper she suddenly became aware of a purring and thumping in her chest There were no other symptoms

On examination the blood pressure was found to be 125/65 mm Hg in both arms The patient was moderately obese She was perfectly comfortable A prominent diastolic thrill was palpable over the entire precordium The heart was enlarged to the left Auscultation was dominated by a very loud cooing murmur which was maximal at the left sternal border but which also was audible over the skull and sacrum

Electrocardiogram revealed the pattern of left axis deviation and left ventricular strain By x ray the aorta was dilated and tortuous and the innominate artery was kinked and dilated Serologic tests for syphilis including a treponemal immobilization test were negative The temperature was always normal and four blood cultures were sterile

Although no signs of congestive failure were present prophylactic digitalization was performed However because of the appearance of numerous ventricular extrasystole digitalis was discontinued

The patient did not return for further observation She had several admissions to another hospital for treatment of congestive heart failure She died at home 21 months after her first hospital admission

The mechanism in this patient may have been cystic medial necrosis as is discussed in the text Also as discussed elsewhere arteriosclerosis is a possible but less likely cause Fenestration (Fig 253) is yet another

confusion with mitral stenosis because of the more rumbling quality (p 149) The fact that it begin immediately with the second sound will help identify it In rare instances an aortic diastolic murmur is louder or even heard only at the apex or in the axilla (the Cole Cecil murmur)

The *shape* of the murmur is usually a decrescendo one beginning essentially with the second heart sound Occasionally especially when musical, it may be crescendo decrescendo in contour the crescendo limb being short but steep Occasionally on auscultation there appears to be a gap between the second sound and the beginning of the murmur (Figs 258 259 and 260) The explanation (860) in some cases can be shown to reside in the brief crescendo which precedes attainment of maximum intensity In other instances there appears to be a true gap (Fig 31 also 1529) In some cases in which AS and left bundle branch block are present (Fig 260) this may be due to paradoxical splitting of the second sound with delayed appearance of the aortic closure sound

A long diastolic murmur may continue straight

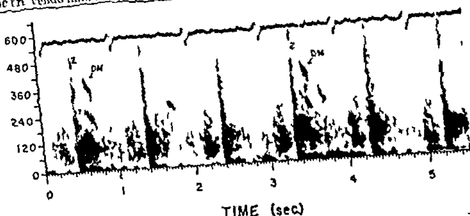


Fig 2[illegible] Highly variable musicality in aortic diastolic murmur of patient with syphilitic aortitis (H R 3 [illegible]) At times there seemed to be a relation to respiration the musical element being greater in expiration

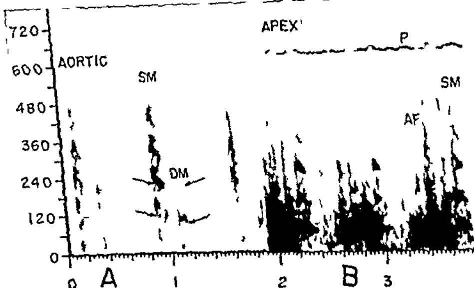


Fig 263 Musical systolic and diastolic murmurs in patient with luetic aortitis J H (F 8890) age 58 The vibrating element may prolapse into the aorta in systole At the apex the vibrating element is less heard and there is both an Austin Flint murmur and a presystolic gallop

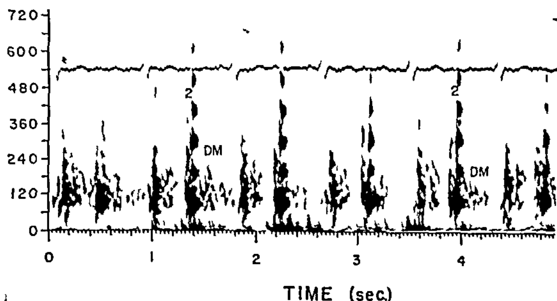


Fig. 269 Circumscribed early diastolic musical element in longer noisy diastolic murmur of aortic regurgitation Syphilitic aortitis in F. D. (616706) colored female age 60 years at time of this recording The patient's clinical course has been surprisingly benign

to the next first heart sound Occasionally the murmur cuts off abruptly at the time of atrial contraction. Presumably atrial contraction raises intraventricular pressure abruptly to a level such that the pressure differential between aorta and pulmonary artery is below the threshold for murmur production More rarely when the regurgitant murmur stops abruptly in mid diastole at a point unrelated to atrial systole there is at its end a sound i.e., a transient which I have termed a reclosure sound These features of the shape of the murmur of aortic regurgitation are displayed in Figure 96

The *quality* of the murmur is usually whiffing, whirring or blowing, depending to some extent on how loud it is On transmission to the apex the quality of the murmur is likely to be altered as a result of selective transmission of those components at frequencies in the range of the natural frequency of the thorax This tends to be at the lower end of the frequency scale The fact that the murmur is lower pitched and more rumbling at the apex does not necessarily imply an origin different from that of the murmur heard at the base

Occasionally the murmur is musical Retroversion of a cusp (Figs 264 to 268) with creation of a lip which is free to vibrate in the regurgitant stream is the classical anatomic basis but fenestration and other less easily described deformities of the cusp may produce a musical murmur Occasionally in so called calcific aortic stenosis there is a musical diastolic murmur as well as the systolic one In such cases the deformed valve orifice functions as the generator of a musical murmur with flow in both the forward and backward direction

The musical murmur with fenestration of a cusp, bacterial endocarditis and valve deformities other than retroverted cusp is rarely as pure as that associated with the latter lesion It usually is a matter of some harmonics in the midst of a noisy murmur Occasionally the murmur of retroverted cusp may disappear being replaced by a conventional noisy aortic diastolic murmur This I have observed at least twice (P. C. 424089 I. S. 103485) In one case a patient with syphilitic aortic regurgitation (F. D. 616706) the musical murmur was very short and limited to the first part of diastole Two years later it had become a more conventional long musical *diastolic* murmur of retroverted cusp

Usually in both rheumatic fever and in syphilis once an aortic diastolic murmur is heard in indubitable form it persists *thereafter* There are reported exceptions however, in both diseases (739)

The systolic murmur of aortic regurgitation i

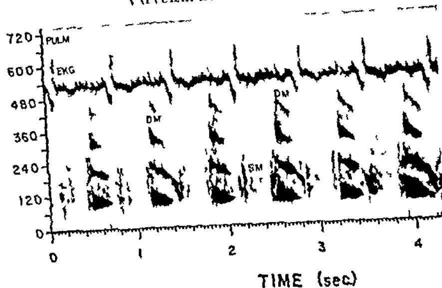


Fig. 270 Same patient as in Fig. 269 two years later. The musical murmur now occupies most of diastole. Pulmonary area. EKG = inverted limb lead 2.

the result of the large stroke volume and the high velocity of ventricular ejection is outlined under Physiologic Considerations. Dilatation of the ascending aorta, a likely accompaniment of aortic regurgitation on the basis of syphilis or of the Marfan syndrome, contributes to the systolic murmur. On rare occasions the systolic murmur may be so intense that a thrill is felt. Leatham (862) observed three such patients with aortic regurgitation due to syphilis. The differentiation of relative aortic stenosis from true organic stenosis is frequently difficult on the basis of the sounds alone. The later the frequency intensity peak of the murmur the more likely is there to be organic stenosis.

The *Austin Flint murmur* of aortic regurgitation is a diastolic murmur at the apex with auscultatory characteristics *resembling* those of the murmur of mitral stenosis (Fig. 262). It is difficult to tell from Flint's *original report (464)* whether the murmur which he characterized as "blubbering" was presystolic or mid-diastolic. *However, in* another publication 24 years later he leaves no doubt that he was discussing a presystolic murmur. It seems that either a mid-diastolic *or a presystolic* apical murmur *may be* associated with pure aortic regurgitation. I would see no reason why the Flint eponym should not be *applied to* both. The change in quality of the primary murmur of aortic regurgitation on transmission to the apex should not occasion *confusion* with the *mid-diastolic* Flint murmur. Also not to be confused are a presystolic gallop or a crescendic S_1 which may occur with aortic regurgitation (977).

When tachycardia is present the erroneous diagnosis of mitral stenosis is especially likely because atrial systole occurs at a time when the factors responsible for a mid-diastolic Flint murmur are operative. Furthermore as with any tachycardia the first sound becomes accentuated to resemble the ringing first sound of MS. An aortic ejection sound occurs commonly in aortic regurgitation and tends to be well transmitted to the apex. The preceding first sound and/or a presystolic Flint murmur may with the ejection sound suggest MS. In the oscillogram too the late ejection sound would be *consistent* with the *delayed* S_1 of MS. In the spectrogram the characteristic features of clicks and snaps (p. 179) are likely to permit *correct identification* of the ejection sound.

A number of theories have been proposed for the genesis *of the Flint* murmur (320). Dilatation of the ventricle probably contributes as in some cases of *relative* mitral stenosis on other bases. Currens and co-workers (320) stated that "left *ventricular* dilatation is apparently not essential for the production of the Flint murmur but may predispose the heart to such a murmur." Actually I doubt that a *Flint* murmur ever occurs without at least slight ventricular dilatation. [illegible]

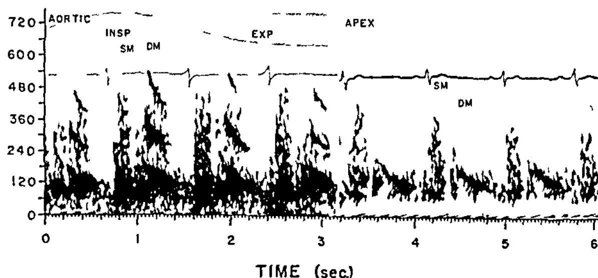


Fig 271 Preferential transmission to apex
Recorded in H I (765151) with musical aortic diastolic murmur audible as far as each olecranon and the lower abdomen and back The intensity and frequency level of the murmur was accentuated in early expiration There is a change in tonality on transmission to the apex caused by preferential transmission of the fundamental

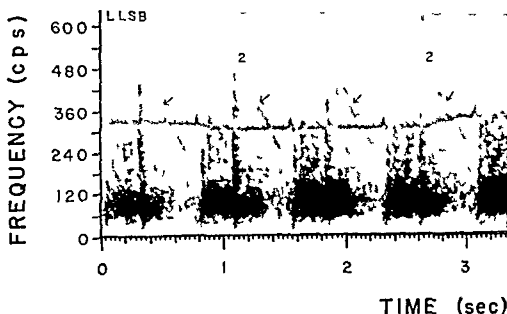


Fig 272 Unusually high pitched musical diastolic murmur in syphilitic disease of aortic valve
LLSB in N G (726022) 50 years old with historical and serologic evidences of syphilis The musical element of his diastolic murmur is unusual for syphilis in that it consists of a single harmonic and has an unusually high pitch exceeding 400 c p s at its peak The harmonic has the typical crescendo decrescendo chevron pattern An early systolic click of dilated aorta introduces a decrescendo systolic murmur

ation of ventricular dilatation is notoriously unreliable the stroke volume is necessarily increased in aortic regurgitation and presumably diastolic volume of the ventricle is also increased Increased flow across the mitral valve may be a contributory factor since relative mitral insufficiency on the basis of ventricular dilatation is to be expected Filling of the ventricle from two sources may result in the mitral cusps' attaining a more nearly closed position at the end of diastole An important factor may be more direct impingement of the regurgitant stream from the aorta on the anterior aortic leaflet of the mitral valve with displacement of the cusp into the stream of blood entering the ventricle from the left atrium This would be expected to produce not only a

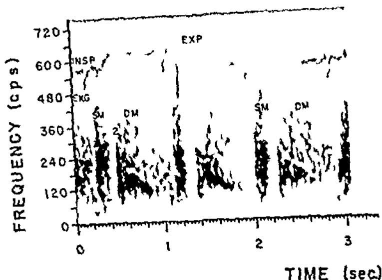


FIG. 73. Musical diastolic murmur with calcific aortic valve disease.

Aortic area in A. F. (73374), 78 year old female who in childhood had growing pains but no definite rheumatic fever. Dyspnea and ankle edema brought her to the hospital. Examination revealed a blood pressure of 210/70/0 mm Hg, kinked carotid on the right, a loud systolic and diastolic murmur both rather musical to the ear, and left ventricular enlargement by x ray and EKG. It was concluded that she suffered from calcific aortic valve disease which was probably atherosclerotic on a rheumatic nidus. The musical elements of the diastolic murmur are well shown and those of the systolic somewhat less well shown.

presystolic but also at times a murmur beginning earlier in diastole following the second sound by a *brief gap. The theory last mentioned has been* supported by most recent writers (e.g. 320) *and was proposed by DaCosta (322) in 1908.* On the basis of ten cases Gouley (581) concluded that there is a characteristic deformity of the right anterior coronary cusp *associated with* Austin Flint murmur, a concave cup shaped deficiency at the inner portion of the leaflet, constituted so as to direct or groove the regurgitant blood toward the lower portion of the anterior mitral curtain. The latter exhibited on its ventricular aspect a variable degree of endocardial thickening which was interpreted as frictional sclerosis.

The *second sound* in aortic regurgitation is probably accentuated in a majority of cases. In mild rheumatic aortic regurgitation fibrosis of the valve is probably responsible and the same *factor* probably operates in more severe cases. In syphilitic aortitis the ringing second sound is well recognized. Another contributing factor in the accentuated aortic second sound may be the systolic collapse phenomenon. More rapid fall in aortic pressure with accelerated aortic valve closure and accentuation of A_2 may occur.

The *peripheral auscultatory signs of* aortic regurgitation are often striking but rarely of specific diagnostic usefulness over and above that of the blood pressure determination and the primary or central auscultatory signs. The main findings of auscultation over peripheral arteries are pistol shot sound, Duroziez's sign and Traube's sign. These have been described earlier (p. 254). The pistol shot sound is a single transient related to the water hammer pulse; the Duroziez sign is a *double* murmur elicited by compression of a peripheral artery with the margin of the bell of the *stethoscope*; the Traube sign consists of two sounds heard without compression of the artery.

MITRAL STENOSIS (MS)

ETIOLOGIC AND ANATOMIC CONSIDERATIONS (1331, 1332, 1333). Rheumatism is virtually the sole cause of true organic mitral stenosis of significant proportion. Congenital mitral stenosis (p. 385) has been recognized with increasing

frequency in recent years, but is still a rarity (456) Trauma was thought to be the cause in one *patient (50), who the authors thought had* sustained a hematoma of the aortic leaflet of the mitral valve

Rheumatic fever causes gluing *together of the* mitral cusps, which themselves become thickened by a fibrosing and sometimes calcifying process The same scarring process involves the chordae tendineae, which may become shortened, if not actually shortened (583), their effective length is reduced by the fact that in the scarring process they become glued together at their cuspal ends and, in effect, incorporated into the cusp Most mitral orifices sufficiently stenosed to give symptoms for which valvulotomy is indicated have a cross sectional area of the order of one square centimeter or less (The normal effective mitral orifice area is of the order of 5.0 cm^2) The end stage is appropriately referred to as a "fish mouth" valve The aortic leaflet of the mitral valve usually *retains enough pliability to play* a role in the mitral opening snap and probably in the snapping quality of the first heart sound Extensive calcification is usually associated with loss of the pliability of the aortic leaflet and with corresponding changes in the auscultatory findings

PHYSIOLOGIC CONSIDERATIONS (576, 579) Physiologic factors are intimately related to the changes in cardiovascular sound in mitral stenosis For example, the level of left atrial pressure or rather the atrioventricular pressure gradient is related to the degree of delay of M_1 and the interval between S_2 and the opening snap, the volume of blood flow as well as the pressure gradient is related to the intensity of the diastolic murmur However these features can be best discussed in relation to the auscultatory changes

Adopting a method commonly employed by hydraulics engineers Gorlin and Gorlin (575) proposed the following formula for estimating effective diastolic mitral valve area (MVA)[3]

$$MVA\ (\text{in cm}^2) = \frac{MVF}{31\sqrt{PC - 5}}$$

where MVF is mitral valve flow (cardiac output per minute of diastolic time) and PC is pulmonary capillary ("wedge") pressure Both of these data can be derived from right heart catheterization The reader will recognize that the above formula is a special case of the following relationship flow is proportional to orifice area and to the square root of the pressure gradient (differential pressure)

$$MVF = J \times MVA \times \sqrt{(\text{left atrial pressure} - \text{left ventricular pressure})}$$

The constant "J" was determined to be 31 Pulmonary capillary pressure is a measure of left atrial pressure Mean diastolic pressure in the left ventricle is assumed to be 5 mm Hg Normally the effective mitral valve area measures approximately 5 cm^2 as calculated from physiologic data applied to the above formula and as checked anatomically

CARDIOVASCULAR SOUND I tell medical students that when they have mastered the auscultatory phenomena of mitral stenosis in all its ramifications they have mastered the entirety of clinical auscultation of the heart This relatively complex subject can be discussed under the following headings

(1) The timing and quality of the first heart sound
(2) The systolic murmur
(3) The second sound
(4) The mitral opening snap
(5) Estimation of the severity of mitral stenosis
(6) The third heart sound
(7) The passive diastolic murmur
(8) The atriosystolic murmur
(9) The pulmonary early systolic click
(10) *The Graham Steell murmur*
(11) Postvalvulotomy changes
(12) Conditions presenting auscultatory signs simulating those of mitral stenosis

The *first heart sound* (mitral closure sound) is delayed and ringing in mitral stenosis The degree of delay bears a direct and rather quantitative relationship to the level of left atrial pressure and therefore to the severity of the mitral obstruction (793, Fig 3) The Q-1 interval in adults is normally between 0.02 and 0.06 sec Leonard *et al* (875) found an average figure of 0.055 sec In individuals with mitral stenosis the interval

[3] As indicated in the footnote on page 263 the Gorlin formula applies only to small or smallish orifices not to the normal orifice

may be as long as 0.12 sec. The pressure in the left atrium is elevated often to values in the range of 35 or 40 mm. Hg in severe cases of mitral stenosis. The mitral closure sound does not occur until pressure in the left ventricle exceeds that in the left atrium. This may take as long as 0.07 sec. after the onset of left ventricular contraction. It will be noted that there are two components to the Q-1 lag, one electrical if you will—the delay between the beginning of the QRS and the onset of ventricular contraction as indicated by the onset of pressure rise, the other mechanical—the delay between the onset of contraction and the closure of the mitral valve when pressure in the left ventricle exceeds that in the atrium. It is the latter which is prolonged in these cases.

Bundle branch block—by prolonging the first phase of the lag—might be expected to confuse the picture and vitiate the usefulness of the Q-1 measurement in gauging the severity of the mitral stenosis. However, the evidence of Braunwald indicates no delay in the onset of contraction in either ventricle in most cases of bundle branch block (p. 166).

It has recently been demonstrated (875) that a prolongation of the Q-1 interval occurs in association with systemic arterial hypertension (see p. 427). Hypertension can therefore interfere with phonocardiographic estimation of *mitral* stenosis in two ways: the manner just mentioned and the effect on the interval between *the second* sound and the opening snap (see below).

In the oscillogram it is the beginning of the first rapid vibrations which are used *in making* the Q-1 measurement. These are almost always also the first large vibrations. There are often some coarse vibrations of small amplitude preceding the mitral closure sound. These were thought to be related in some manner to atrial systole until it was noted (308) that they persist in cases of atrial fibrillation. They may represent the elusive and much debated muscular (i.e., myocardial) contribution to the first heart sound. It is not surprising that they precede the valve closure sound. Occasionally, especially in the spectral phonocardiogram, one sees a discrete sound just preceding the sharp mitral closure sound which satisfies the criteria for a tricuspid closure sound (Fig. 281). That this is not more often seen in mitral stenosis is probably related to the fact that normally mitral closure slightly precedes tricuspid closure. Considerable delay of the mitral closure sound is necessary before separation of the two becomes evident through a paradoxical sequence of closure. Hultgren (726) has demonstrated a sound preceding the mitral closure sound and having characteristics one might predict for a tricuspid closure sound. Specifically, its intensity varied inversely with the duration of the preceding diastole in cases of atrial fibrillation. The sound was still present when recordings were made from the exposed heart at surgery; impact against the thoracic cage is not its mechanism. Some reported examples of so interpreted presystolic murmur persisting after the development of atrial fibrillation (739, 1188) were probably cases of a tricuspid sound preceding the mitral closure sound.

With extensive calcification of the mitral valve the first sound may be diminished even though tight *mitral stenosis* is present and is the predominant lesion. (Furthermore an opening snap may be absent in such cases.) The intensified ringing M_1 persists after mitral commissurotomy unless a good deal of mitral regurgitation has been inadvertently produced.

The oscillogram displays the unusually great intensity of the mitral closure sound and the SPCG displays this plus the frequency characteristics responsible for its ringing quality to the ear. A ringing heart sound is to ordinary noisy heart sound as a musical murmur is to noisy murmurs. *In the SPCG the ringing* M_1 of mitral stenosis tends to display harmonic pattern just as do musical murmurs. Furthermore it has components of a higher frequency than normally and of course its over-all intensity is increased.

The great intensity and the particular quality of the first sound in mitral stenosis are the result mainly of the fibrotic change which has taken place in the mitral valve curtain, especially the aortic or antero-septal leaflet. Anatomically it is impossible to imagine much sound being produced in coaptation of the margins of the cusps which are relatively close together. Sudden tensing of the shortened, stiffened chordae tendineae may contribute. Observations on the first heart sound in mitral stenosis appear to shed light on two important physiologic features of the normal heart sounds and normal valve func

tion (1) the first sound is related, in the main, to AV valve closure, (2) valve closure sounds are produced more by tensing or snapping of the belly of the valve cusps than by collision of the coapsing valve margins

Normally with atrial fibrillation alone there is variability of the intensity of S_1 as indicated in Figure 447 (p 437) at values of diastole between 0 20 and 0 25 sec the intensity reaches a minimum, at still longer diastolic periods there may be a secondary increase in intensity According to Ravin and Bershof (1247) in severe mitral stenosis there may be essentially no variation in

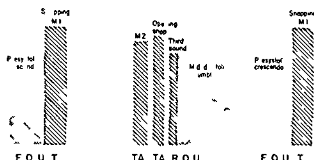


FIG 274 Duroziez s fout ta ta rou of pure mitral stenosis

The sound marked third sound is usually little more than a concentration of the first part of the mid diastolic murmur in those cases in which the rumble does not begin immediately with the opening snap

the intensity of S_1 In a certain number of patients, intermediate as to severity (this category was represented by four out of ten patients studied) there is a slow decline in intensity reaching a minimum value at diastolic periods of 0 5 to 0 7 sec The explanation offered for the finding in the normal situation is that as ventricular filling progresses the valvelets "float up" to a semi closed position and make less noise in closing In mitral stenosis diastolic filling is delayed and prolonged the normal process of 'floating up' takes a longer period or does not occur at all because of elevated left atrial pressure In Figure 282 is presented a case of mitral stenosis and atrial fibrillation in which S_1 varies in intensity depending on the length of the preceding diastolic period

A *systolic murmur* may occur at the apex if there is mitral regurgitation of any degree It was formerly thought that for practical purposes mitral stenosis never exists in the absence of some degree of mitral regurgitation That this is not true is one of the many facts revealed in connection with mitral valve surgery So called "pure" mitral stenosis does exist Even though the stenotic orifice is rather rigid the cusp curtain may be sufficiently pliant to appose above the orifice (408) The mitral mechanism then becomes *essentially a flutter valve* A *systolic murmur* is sometimes heard at the apex in cases of mitral

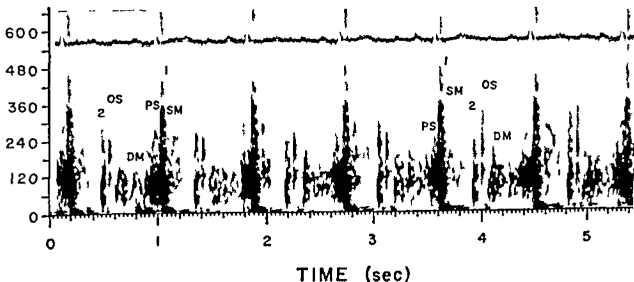


FIG 275 Apex in D C (687240) with predominant *mitral stenosis* Note the close reproduction of the Duroziez device shown in Figure 274

tenosis yet no regurgitant jet is found at operation. Assuming that the heart is at the time beating as vigorously as before thoracotomy or at least sufficiently vigorously for regurgitation to be expected—operationally speaking, if the blood pressure is in the usual range—then the basis for the systolic murmur in many of these cases may be relative tricuspid insufficiency caused by dilatation of the right ventricle rather than mitral regurgitation (483, 1307, 1482). Accentuation of the systolic murmur with inspiration helps in this identification but unfortunately is not always present. The murmur of tricuspid regurgitation is likely to be heard unusually far to the left in this situation because of the dilation of the right ventricle and clockwise rotation of the heart.

Mounsey and Brigden (1126) remarked on the frequent occurrence of a short decrescendo early systolic murmur at the apex limited to the first part of systole and not related in any consistent manner to the finding of mitral regurgitation at operation. This murmur is illustrated in several of the recordings presented here, e.g., *Figure 273*. This murmur, unlike the murmur of the usual mitral regurgitation, is not holosystolic.

There may be a short, not loud, early systolic murmur in the pulmonary area *and* just below. The site of origin is the dilated pulmonary artery and the murmur is often introduced by an early systolic click (see later).

Systole in cases of mitral stenosis is often impressively short. Even on auscultation the second sound frequently seems to come very soon after the first sound and the principal finding is a long diastolic rumble ending in a sharp first sound. The interval between the two heart sounds is

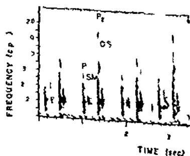


Fig. 276. Pulmonary area in pure mitral stenosis with sinus rhythm. Opening snap louder here than at P₂.

shortened because of delay in the first heart sound. In addition there probably is a true abbreviation of systole due to the small stroke volume.

The *second sound* in the pulmonary area in mitral stenosis is usually accentuated as a result of pulmonary arterial hypertension. In pure mitral stenosis the heart sounds in the aortic area, especially the second sound, are usually strikingly faint, especially in comparison with the loud P_2 and ringing M_1. This is Skoda's sign—loud P_2, faint A_2. The faint aortic sound undoubtedly is related in part to the relatively low aortic pressure in cases of pure mitral stenosis, but also to clockwise rotation of the heart, which occurs in these cases and tends to swing the base of the aorta to the left and closer to the left sternal border. The clockwise rotation of the heart results in considerable part from the enlargement to the right of the left atrium which lies on the posterior aspect of the heart. When A_2 is loud in combination with signs of mitral stenosis, one must suspect the presence of aortic valve changes, specifically fibrosis. When A_2 is loud, a diastolic murmur at the left sternal border can be suspected as representing aortic regurgitation rather than a Graham Steell phenomenon.

Usually at the apex too the second heart sound is faint in mitral stenosis. This is because the aortic closure sound is normally responsible for S_2 at the apex. With pronounced accentuation the pulmonary closure sound may be transmitted to the apex, the clockwise rotation of the heart with the result that the right ventricle constitutes the apex contributes to this transmission.

Splitting of S_2 does not, contrary to a prevalent impression, occur to an impressive degree in mitral stenosis. The impression that it does has been created by the fact that the opening snap is more often than not clearly audible in the pulmonary area. True *splitting of S_2* is more a feature of mitral regurgitation.

The *mitral opening snap* (OS) is the closest approximation to a unique and pathognomonic auscultatory sign in mitral stenosis. It immediately follows the second heart sound, being separated from the aortic closure sound by an interval dependent on the level of atrial pressure and therefore the grade of mitral stenosis. The S_2-OS measurement is then a gauge of the

severity of the mitral stenosis, just as is the Q-1 measurement. The S_2-OS interval may be as little as 0.05 sec. in severe cases of mitral stenosis. In mild cases, especially cases of mild stenosis in association with systemic arterial hypertension (see Fig. 289), the S_2-OS interval may be as long as 0.14 seconds. After mitral commissurotomy, the opening snap persists more often than not and the S_2-OS interval lengthens to values of as much as 0.14 sec. if satisfactory relief of the mitral obstruction has been achieved.

The interval between S_2 and OS varies in atrial fibrillation, depending on the duration of the preceding diastolic period (Fig. 278). With longer diastolic periods there is a longer time for decompression of the atrium and the next OS occurs later with a wider S_2-OS interval. The converse is true with short diastolic periods. In

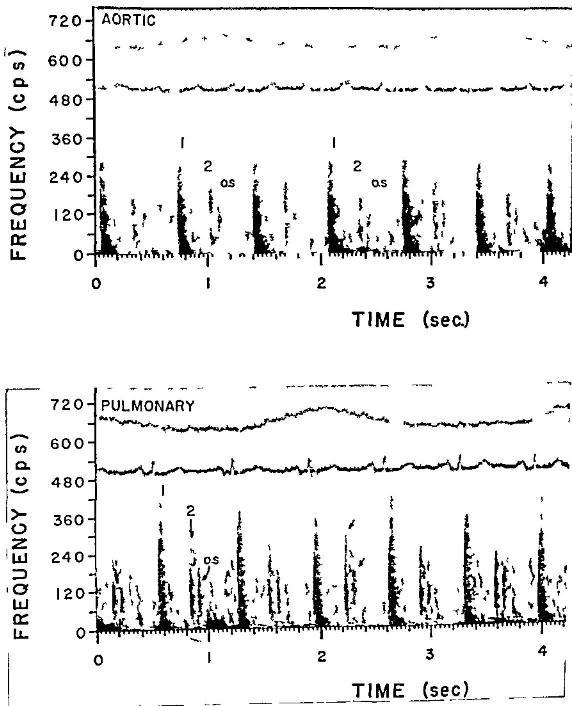
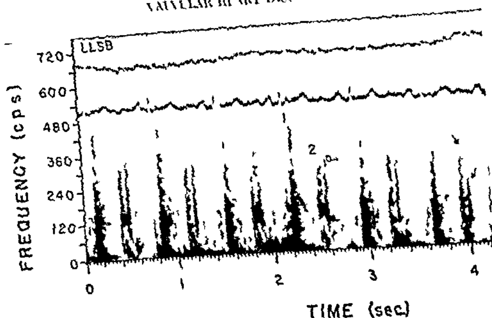


Fig. 277. A (*upper*) and B (*lower*). See legend with Figures 277 C and D.



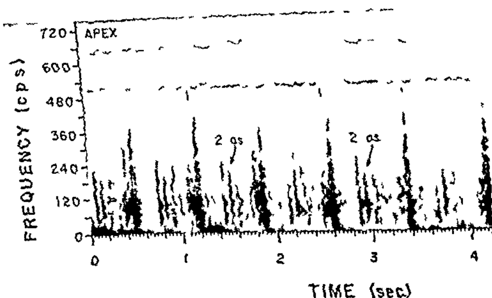


Fig. [illegible] *B*. Typical rheumatic mitral stenosis.

In the tracing of respiratory phase at the top of each recording, inspiratory movement is upward and expiratory movement is downward, as in all other recordings in this [illegible]. In the aortic area the heart sounds are characteristically [illegible] accentuated second sound would suggest rheumatic [illegible] valve. The first sound is more striking than usual; its delay relative to the QRS indicates that it is the mitral closure sound heard in the aortic area with unusual clarity because of its accentuation. No splitting of [illegible] demonstrated; only aortic closure is represented. The opening snap (OS) is well demonstrated in the aortic as in other areas. In the pulmonary area [illegible] and the second sound is split, especially during inspiration (see the upper arrow in the [illegible] of the fourth [illegible] in B). The simultaneous demonstration of a split second sound and the opening snap leaves little doubt of the identity of the [illegible] sounds. There is probably little pulmonary hypertension in this case. The split second sound and opening snap are similarly well demonstrated at the lower left sternal border. At the apex the mitral [illegible] is accentuated and delayed. The second sound is unitary and [illegible] normally the case [illegible] the result of [illegible] valve closure. The diastolic rumble begins immediately with the opening snap [illegible] decrescendo, then [illegible] in presystole.

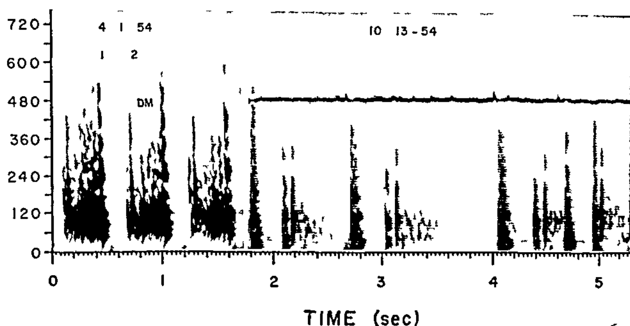


Fig 278 Effect of atrial fibrillation in mitral stenosis

Apex in S O N (616116) before and after development of atrial fibrillation The first recording has a diastolic rumble beginning immediately with the opening snap With development of fibrillation the presystolic element disappeared In the first recording the S_2 OS interval is short because of the rapid heart rate and short diastolic periods In the second record it is longer and varies directly with the length of the preceding diastolic period Q S_1 delay is also demonstrated

the upright position the S OS interval is longer than in recumbency (1364, 1454) Mounsey (1124) found no respiratory variation in the interval but Schoelmerich and Gehl (1364) did

Well (1525 1527) found a rough correlation between size of the mitral orifice as discovered at operation and both the Q 1 interval and the S OS interval The correlation was much improved by relating Q 1 minus S OS to mitral size (Both values were corrected to a cycle length of 0 8 sec) He thought the improved correlation resulted from a cancelling of the effect of varying amounts of fibrosis and calcification of the valve possibly fibrosis and calcification would increase both the Q 1 and the S OS interval Also the two measurements together might he thought reflect mean diastolic gradient more accurately The study of Bayer Wolter *et al* (64, 66 1585) correlating pulmonary capillary pressure with S OS interval seems to indicate that fibrosis and calcification in fact have no important effect per se—pressure differential is the only factor of importance in determining variation in the intervals in question (The S OS interval was the one specifically studied by Bayer s group [see p 190]) Wells failure to demonstrate a closer correlation in the first instance might have been the result of the inaccuracies of the measurement of the mitral valve by palpation at surgery Furthermore taking only valve size into account and not flow—the other factor in determining left atrial pressure—would not be expected to result in perfect correlation However when correlations with the pressure gradient at operation were attempted he again found that the best correlation was provided by the index of corrected Q 1 minus corrected S OS

Julian and Davies (761) could find no satisfactory correlation of the Q 1 interval with pressure in the left atrium although the S OS interval correlated well They suggested that the lack of correlation of the first was because of the relatively wide range of values in normals (0 02 to 0 06 sec , according to Kelly (778)) Difficulties in obtaining a satisfactory correlation of grade of mitral stenosis with the Q 1 and S OS interval may have been due in some instances to uncertainties in the identification of the mitral closure sound and the opening snap in the oscillogram

Kuo and Schnabel (827) have presented evidence that when hemodynamically significant mitral regurgitation or aortic regurgitation is

present variation in the Q 1 and S₂-OS interval depending on the length of the preceding diastolic period does not occur

The opening snap is, as its name suggests, a dry snapping sound as a rule However this is not always definite to the ear Furthermore the opening snap is situated so close to S₂ in cases of severe mitral obstruction that the S₂ OS combination in the past has been frequently misinterpreted as a split second sound specifically a split I The confusion is compounded by the fact that contrary to what might on first thought be considered likely the mitral opening snap is well heard—in fact usually better heard than at the apex—at the left sternal border and pulmonary area and even in the aortic area It can be detected in the suprasternal notch and the right base of the neck in some instances Tain (912 913 917) has pointed out that whereas the mitral opening snap is usually audible in the suprasternal notch a split second sound is not—a possibly helpful differential point

In the oscillogram there is nothing particularly unusual about the opening snap as compared with other transients In the SPCG the opening snap usually has the features which characterize snaps and clicks in general and permit its differentiation from a valve closure sound (1) brief duration (2) a frequency bottom which does not come to zero (3) a tendency to pure frequency content

Wood (1088) found that the opening snap was absent in a case of predominant mitral stenosis in which aortic regurgitation was also present He suggested that the regurgitation against the aortic leaflet of the mitral valve during isometric relaxation and early diastole might prevent the sudden billowing of the mitral sail responsible for the snap Kelly and Tain (912) in one case and Mozer and Duchosal (1128) in two failed likewise to detect an opening snap when aortic regurgitation was present On the other hand Margolies and Wolferth (1010) found five cases of opening snap in spite of associated aortic regurgitation

Often the presystolic diastolic murmur does not

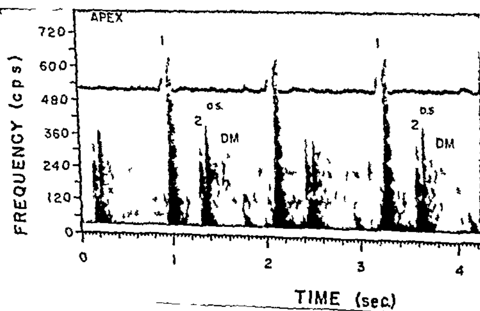


Fig. 99 Rheumatic mitral stenosis with unusually high pitched diastolic murmur
In this recording from the apex the appearance of the characteristic snapping mitral first sound is displayed It is louder than normal has a greater frequency span and has a conspicuous harmonic pattern This the mitral closure sound is delayed relative to the QRS It is followed by a striking opening snap The opening snap is followed immediately by a diastolic murmur of appreciably higher pitch than is usually found in mitral stenosis All features of this case are typical of mitral stenosis of moderately severe degree That the murmur in fact had its origin at the obstructed mitral orifice is supported by its disappearance after mitral valvulotomy In spite of the presence of sinus rhythm no presystolic component of the diastolic murmur is demonstrated in this recording Electrical interference at 120 cycles and to some extent at 240 cycles is present

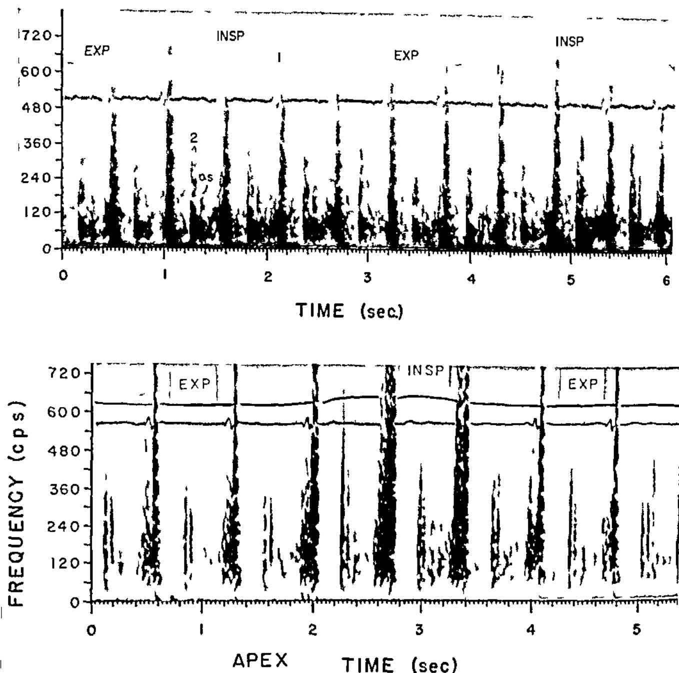


Fig. 280 Changes in the diastolic murmur of mitral stenosis with respiration

(*Above*) with inspiration the second sound becomes split and it is then the second component (pulmonary valve closure) which dominates. The fact that pulmonary valve closure is so well heard at the apex indicates considerable pulmonary hypertension. The diastolic rumble is intensified early in *expiration*.

(*Below*) apex in M. W. (618373) 33 years old and asymptomatic. The presystolic murmur is accentuated in inspiration and early expiration. S_2-OS interval is shortest during cycles with loudest diastolic murmur. All clinical and radiologic evidences point to mitral stenosis not tricuspid stenosis in this patient.

begin immediately with the opening snap but only after a brief interval. In some of such cases there is at the beginning of the murmur a concentration of vibrations which should be considered a sound. Since it has the temporal relationship of a *third heart sound*, I will so refer to it. It is difficult to imagine there being a rapid filling sound arising in the left ventricle in MS in view of the impediment to ventricular filling. It may be a third sound produced in the right ventricle (1588, 1062). Contro (285) found a third heart sound in 5 of 84 cases of presumed mitral stenosis

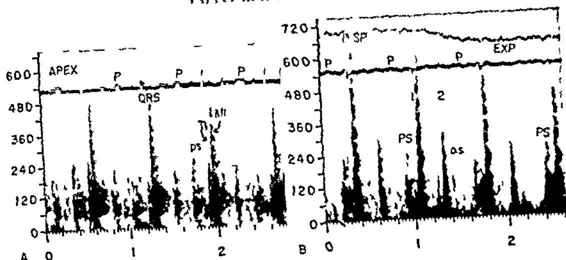


Fig 281 Rheumatic mitral stenosis with prolonged atrioventricular conduction
Fundamentally the presystolic murmur of mitral stenosis is the same as the systolic murmur of aortic or pulmonary stenosis All are ejection stenosis murmurs In the case of the presystolic murmur of mitral stenosis the typical Christmas tree pattern is cut short by the snapping first sound when the atrioventricular interval is of normal duration When the atrioventricular interval is prolonged as in the cases the typical pattern becomes evident (*At left*) the first sound is split Tricuspid closure occurs in its normal relation to the QRS Mitral valve closure which normally occurs slightly earlier than or coincident with tricuspid closure is delayed and accentuated Usually in mitral stenosis the presystolic murmur obscures the tricuspid closure sound which becomes evident only in cases such as this or cases of atrial fibrillation (At right) the same Christmas tree pattern of the presystolic murmur is demonstrated because of PR prolongation This recording was made after mitral valvulotomy A faint opening snap persists

One of the five in fact had pure mitral regurgitation The other four had tricuspid regurgitation in association with mitral stenosis Contro concluded that the right ventricle was the site of origin of the third sound

What the difference is between cases in which the early diastolic murmur begins immediately with the opening snap and those in which it begins only after a brief interval is not clear Although there might be an anatomical basis the difference more likely is quantitative It is in severe cases that the murmur begins with the opening snap

The *passive diastolic murmur* is rumbling in quality With long diastolic period it is likely to display a somewhat decrescendo character It is the only part of the diastolic murmur which persists when atrial fibrillation supervenes As a variation on the usual rumbling quality White (1338 p 97) states Rarely the murmur of mitral stenosis may be a gentle and moderately high pitched blow We have observed two or three such patients (see Figs 279 and 282) In one the murmur had a rather scratchy quality The intensity and duration of the murmur are more reliable indices of the severity of mitral stenosis (1062)

The *presystolic* or better *atriosystolic* murmur

In the 1890s Raymond Tripier and Devic and also Brockbank (see p ...) raised the question of whether the so called presystolic murmur may not really be protosystolic The question was reopened by Callavardin (10) and by Nichols and colleagues (1149) Callavardin was puzzled by the fact that they did not find as they would expect a pause even a small one between the atriosystolic murmur and S Nichol group thought that at surgery the palpating finger in the left atrium could feel the vibration of the stenotic mitral diaphragm as it billowed toward the atrium with early ventricular contraction and that the vibration corresponded to the so called presystolic murmur which could be more accurately called protosystolic Callavardin (610) suggested early mitral regurgitation to account for the obliteration of the gap between the atriosystolic murmur and the snapping S

It is true that with a PR interval of normal length the presystolic murmur continues into electrical systole of the ventricle i.e. it continues until well after the onset of the QRS the delay in S increases this early systolic extension of the murmur Both the Nichol and

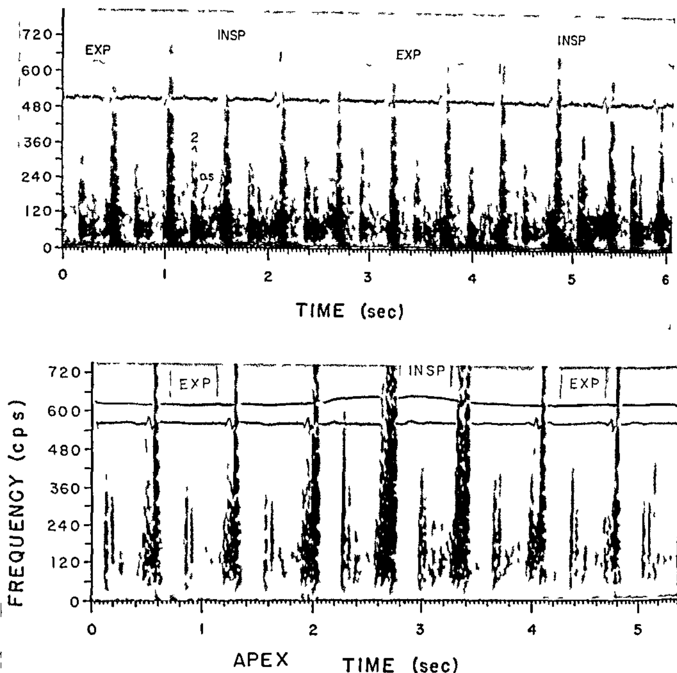


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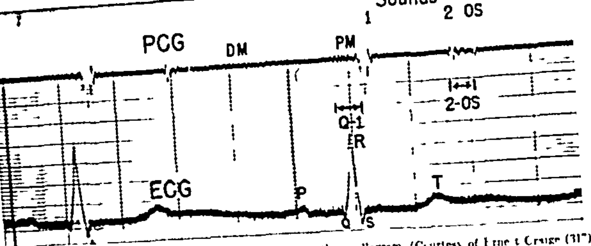


FIG 283 Measurement of the Q 1 and 2 OS interval from the oscillogram (Courtesy of Ernest Craige (317) and the *New England Journal of Medicine*)

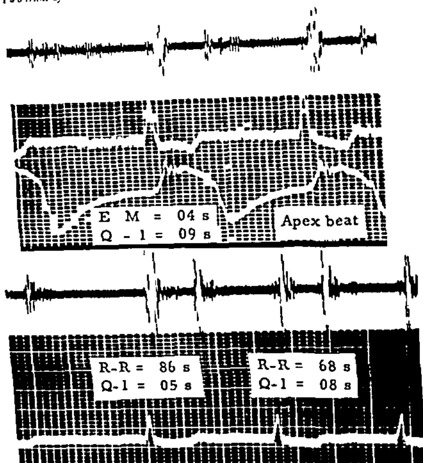


FIG 284 Measurement of Q 1 interval from the oscillogram (Courtesy of Kelly (779) and *Circulation*) (*top*) Record obtained on a middle aged man with proved mitral stenosis The electric mechanical interval (EM) is demonstrated to be 0 04 sec by the systolic deflection of the apex impulse This was confirmed by direct measurement of ventricular pressure at operation In the absence of mitral stenosis the first sound occurs at this time In this instance the first sound begins 0 09 sec after the beginning of the QRS Note the deflections of the apex beat simultaneous with the first sound (*bottom*) Trace obtained from subject with proved mitral stenosis Atrial fibrillation with varying RP interval is present When the preceding diastole is long the Q 1 is short and when diastole is short the Q 1 lengthens A long diastole allows better left atrial emptying and consequently a lowering of the left atrial pressure

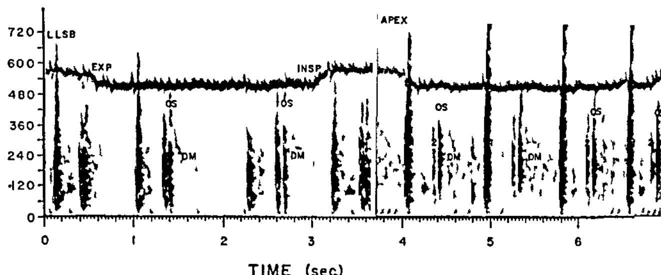


FIG. 282 Mitral stenosis with atrial fibrillation

LLSB (*left*) and apex (*right*) in P. M. (742071), 48 year old female. The S_2-OS interval is longer after longer diastolic periods. S_1 is less loud after longer diastolic periods. These features are not evident at the apex where the rate was more regular during the period of the recording. A peculiar musical murmur in systole may be of pericardial origin. The diastolic murmur is unusually high pitched for mitral stenosis. Even the diastolic murmur at LLSB is probably of mitral origin.

is essentially an ejection stenosis murmur, and like that of aortic stenosis is diamond shaped and Christmas tree shaped in the oscillogram and SPCG respectively. However when PR is of normal duration and atrial systole bears a normal relationship to ventricular systole, the Christmas tree is as it were cut in half vertically and the result is the so called presystolic crescendo. When the PR interval is prolonged the full configuration of the atriosystolic murmur is revealed (Fig. 281). Creighton Bramwell (1[illegible]) pointed out that in such cases if the atrioventricular relationships are such that atrial systole falls in early diastole the atriosystolic murmur is louder than otherwise. This is true probably because atrioventricular pressure gradient is greater at that time (pressure in the ventricle is lowest), atrial contraction is by Starling's law most forceful because the volume of blood contained in the atrium is greatest, and the passive diastolic and atriosystolic murmurs are summated. The presystolic murmur disappears with the advent of atrial fibrillation. At times with atrial fibrillation and a rapid ventricular rate there appears to be a presystolic crescendo, but this is only an illusion created by the fact that the passive murmur continues right up to the snapping M_1. Some, beginning particularly with Lewis (904), have in fact claimed that the crescendo character of the presystolic murmur is always an auditory illusion. They are led to this conclusion by the fact that the oscillographic PCG often shows no crescendo of intensity, possibly because of inadequate frequency response characteristics. The spectral PCG leaves no doubt that the crescendo is *bona fide*.

The two parts of the diastolic murmur are best

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the Gallavardin theories seem unlikely, however; the billowing of the mitral curtain toward the atrium with ventricular contraction would be expected to be an abrupt snapping movement. Mitral regurgitation cannot be expected to precede the first sound because if there is a reversal of the polarity of atrioventricular pressure differential to produce regurgitation, the reversal will produce the first heart sound. Either the Gallavardin or the Nichols theory would require that with atrial fibrillation some vibrations would persist and be recorded between the onset of the QRS and S_1. Such vibrations are not recorded; at least the only vibrations which are recorded have an appearance consistent with an origin in closure of the tricuspid valve or in the contracting myocardium itself.

The lack of gap between the presystolic murmur and the first sound is perhaps not troublesome if one thinks of the presystolic murmur as one part of a bisected ejection stenosis murmur (i.e., half a Christmas tree or diamond). The rising pitch which Brockbank (p. 2[illegible]) felt was so difficult of explanation on the basis of an atriosystolic murmur is likewise accounted for.



Fig 286 From top to bottom pressure curve in aorta left ventricle and left atrium recorded simultaneously with equipotentiative manometer and on the same base line (*Left*) Subject with normal circulation (*Right*) Subject with mitral stenosis (1) Onset ventricular contraction (X) closure of mitral valve in MS (2) opening of aortic valve (1-2) isometric contraction (2-3) maximum ejection (3-4) reduced ejection (5) aortic valve closure (5-6) isometric relaxation (6) opening of mitral valve (6-7) rapid filling (7-8) diastasis (8-9) atrial systole

The large diastolic gradient in mitral stenosis is evident as is the reason for the delayed M1 and abbreviated S_2-OS interval In connection with genesis of S_4 it is of interest that in the normal recordings atrial pressure falls below ventricular pressure at the point in the heart cycle when S_4 occurs (Courtesy of Braunwald *et al* (169) and *Circulation*)

composition the murmur is best heard with the *bell* chest piece *applied lightly* to avoid the diaphragm effect of the underlying skin

Careful search for a diastolic murmur is indicated when one hears a snapping first heart sound and or an opening snap both of which are rather frequently present in the absence of a diastolic murmur on routine auscultation The presystolic portion of the diastolic murmur is the one most likely to be present when there is sinus rhythm and when it is a matter of only one being heard Although passive flow across the valve may not have sufficient velocity to generate a murmur the contraction of the atrium with active propulsion of blood through the obstruction especially because left atrial hypertrophy is usually present produces sufficiently rapid mitral flow to result in a murmur

Duroziez (392) invented the onomatopoetic device fout ta ta rou for the sounds at the apex in pure mitral stenosis The significance of each component of the device is indicated in Figure 274 The actual imitative usefulness of Duroziez s device is destroyed if too anglicized a rendition is provided fout must be pronounced by expelling air at unusual high velocity past the tongue and through the lips and teeth ta ta is given a staccato or machine gun tempo rou has a rolling r Furthermore someone has referred to mitral stenosis as resulting in the bowling alley heart the second sound and opening snap correspond to the impact of the ball

heard at the apex, often in a very confined area. However, occasionally in young subjects with small, thin chests and a loud diastolic murmur of mitral stenosis the murmur will be heard unusually widely, sometimes even in the aortic area, but in such instances the point of maximum audibility is still the cardiac apex.

The intensity of the murmur is dependent in part on the volume and, therefore, on the velocity of flow across the stenotic valve. When the patient is in congestive heart failure no murmur may be heard because of low flow. When severe pulmonary vascular disease develops the murmur may be considerably diminished. Or the association of pulmonary vascular disease on some other basis may obscure the diagnosis because of minimal or absent diastolic murmur. For instance, in patients with severe chronic bronchial asthma and patients with severe pulmonary emphysema the presence of mitral stenosis may be long overlooked. The presence of emphysema in such cases impedes

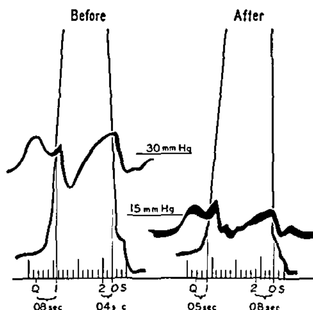


FIG. 285. Relation of left atrial pressure to Q 1 and 2 OS intervals.

Pressure recorded during operation from the left atrium and the ventricle in a patient with severe mitral stenosis. Q refers to the beginning of the QRS complex of the electrocardiogram; 1, to the first rapid vibration of the first sound; *2*, *to the second sound*; *OS*, *to the* opening snap of the mitral valve. Phonocardiograms were taken preoperatively and postoperatively when the heart rates were similar. (Courtesy of Kelly (779) and *Circulation*.)

transmission of any murmur which might be produced. Incidentally, it is of further interest that the murmur may be heard only at an unusually low site, e.g., the left costal margin in the midclavicular line in such cases. Among a series of mitral stenosis with very high pulmonary vascular resistance reported by MacKinnon, Wade and Vickers (1013), there was no diastolic murmur in three. Rawn (1248) described an interesting patient with myxedema and mitral stenosis in whom the latter diagnosis was not made until the myxedema was treated and mitral flow increased.

Levine and Love (890) described 19 cases of mitral stenosis in which no murmur was present at one time or another. "True and totally mute mitral stenosis" must be very rare; rather it is usually the physician who is deaf. In the absence of a diastolic murmur the opening snap and the telltale change in the mitral first sound persist.

Cognizance is taken of the role of flow in the genesis of the murmurs of mitral stenosis by the several maneuvers (exercise, amyl nitrite inhalation (888), etc.) employed to bring it out, all of which *are designed to increase flow*. *Since the* patient with severe mitral stenosis cannot increase his stroke volume—and his mitral flow—much of the effect of these measures is largely mediated through the tachycardia. The shortening of diastole creates the necessity for more blood to pass through the orifice in a given unit of time. With sinus rhythm there is to some extent a summation of the effects of passive flow with those of atrial systole.

The murmur of mitral stenosis is loudest in the recumbent position, especially in the left lateral decubitus, which probably brings the apex more intimately in contact with the anterior chest wall and for that reason favors transmission *of the* murmur. The act of turning into the left lateral decubitus many times is sufficient exertion to bring out the murmur in the first few of the cardiac cycles which follow. The usual practice is to have the patient exercise by sitting up and lying down several times in rapid succession. He then turns immediately *into the left lateral* decubitus, suspending respiration if possible, and the physician listens attentively for the telltale *murmur*. Because of its relatively low frequency



Fig 28. From top to bottom pressure curve in aorta left ventricle and left atrium recorded simultaneously with equisensitive manometers and on the same base line (*Left*) Subject with normal circulation (*Right*) Subject with mitral stenosis (1) Onset ventricular contraction (X) closure of mitral valve in MS (2) opening of aortic valve (1 2) isometric contraction (2-3) maximum ejection (3 4) reduced ejection (4) aortic valve closure (5-6) isometric relaxation (6) opening of mitral valve (6-7) rapid filling (7-8) diastasis (8 9) atrial systole

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composition the murmur is best heard with the *bell* chest piece *applied lightly* to avoid a diaphragm effect of the underlying skin

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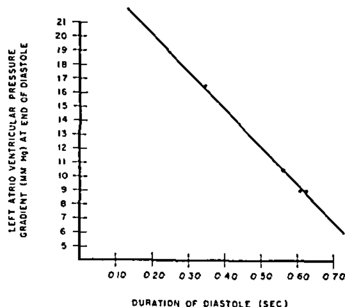


FIG 287 Relation between left atrioventricular gradient at end of diastole and duration of diastole in patient with mitral stenosis and atrial fibrillation The greater the gradient the greater the Q 1 interval can be expected to be The gradient was measured from records similar to that in Figure 286B however the patient had atrial fibrillation (Courtesy of Braunwald et al (169) and *Circulation*)

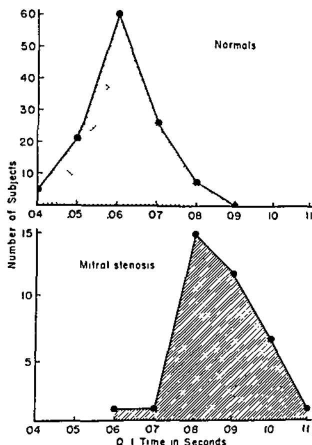


FIG 288 Q 1 time in patients with mitral stenosis compared with that in normal subjects (Courtesy of *Craige (317) and the New England Journal of Medicine*)

on the floor and one bounce the rumble corresponds to the sound of the ball rolling down the alley, the snapping first sound is the impact of the ball on the pins

An early systolic click generated in the dilated pulmonary artery is frequent in mitral stenosis It is loudest in the pulmonary area and may introduce a short murmur This sound (p 129) may be produced by snapping of the arterial wall under the impact of ventricular ejection The click usually occurs about 0 09 sec after the beginning of the QRS of the electrocardiogram The click occurs with dilation of the pulmonary artery due to other causes In the oscillogram and to a much lesser extent to the ear, the systolic click can be misinterpreted as a delayed snapping first heart sound For example, in a patient with primary pulmonary hypertension or multiple emboli the radiologic findings may suggest mitral stenosis and the early systolic click may be taken for a snapping delayed mitral first sound

When pulmonary hypertension is severe a Graham Steell murmur of pulmonary regurgitation may be present It is heard at the left sternal border is seldom loud, and at times (rarely in fact) shows accentuation with inspiration Differentiation from the murmur of mild aortic regurgitation is usually difficult since peripheral signs of aortic regurgitation may be absent Accentuation of the second sound in the aortic area suggests aortic valve disease (see p 172) and if the diastolic murmur is audible at all at the right of the sternum the murmur is probably of aortic origin (652)

After commissurotomy (281 519 1139) the diastolic murmur is diminished as a rule and occasionally is completely abolished During the week or ten days immediately following operation the impression may be obtained that the murmur has completely disappeared However when the patient becomes more active with increase in cardiac output and therefore in mitral flow the murmur is found to be still present The first sound

remains snapping (1425) unless a great deal of mitral regurgitation has been produced but the Q-1 delay is reduced Likewise the opening snap usually persists but the S_2-OS interval is increased P_2 may be diminished in intensity and a Graham Steell murmur may disappear Occasionally an associated valve lesion which was not suspected before operation or was deemed insignificant blossoms forth after relief of the mitral obstruction Specifically the auscultatory signs of aortic stenosis may become clearer (Fig 300)—the patient may fare poorly after mitral valvulotomy if the aortic obstruction is not also relieved—and those of tricuspid stenosis (Fig 320) may also to some extent be accentuated After operation there may be both an atrial gallop and presystolic murmur recorded at the apex Atrial gallop alone have occurred frequently The atrial sound sometimes has the characteristics of a snap (see Figure 297) Production of such a presystolic snap through collaboration of the left atrial hypertrophy and residual fibrosis of the mitral mechanism may perhaps not surprising The sound may disappear late after operation Atrial pleuropericardial adhesions cannot be excluded as a mechanism for the sound

Phonocardiograms before and after mitral valve surgery are not only within the abilities of every institution in which valve surgery is *done* but are in my opinion absolutely mandatory in the minimum pre- and post-operative evaluation Repeated phonocardiographic observation with attention to the indices of *severity of mitral* stenosis constitute one of the best *methods of* postoperative detection of restenosis (1072) (Fig 273)

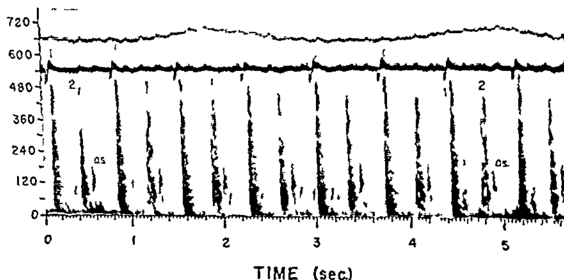


FIG 289 The influence of atrioventricular pressure gradient on S_2-OS interval The upper recording is from the pulmonary area of a *patient with* severe arterial hypertension and a mild degree of mitral stenosis (Upper line = respiration mark with upward motion indicating inspiration downward motion *indicating* expiration) The second sound becomes split with *inspiration* In spite of the systemic hypertension the pulmonary closure is loud and predominates Of particular note is the unusually great S_2-OS interval measuring 0.14 sec from beginning of S_2 to beginning of opening snap in most cycles *Other* clinical evidence indicates only a low grade of mitral *obstruction in this case* A second factor in the S_2-OS prolongation in this *particular case* the systemic hypertension The patient's blood pressure was 220/140 *mm* Hg at the time of this recording suggesting that aortic valve closure might *occur perhaps* at 150 mm Hg and that an appreciably longer time will be required for intraventricular pressure to fall to the level of intra atrial pressure at which time the opening snap will occur These considerations are graphically *presented* in the graph on the next page (Fig 289 *B*)

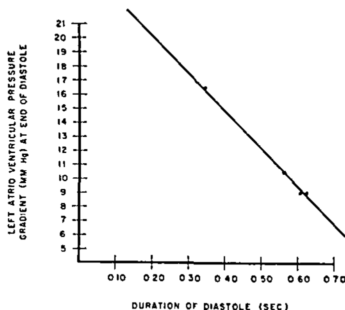


FIG 287 Relation between left atrioventricular gradient at end of diastole and duration of diastole in patient with mitral stenosis and atrial fibrillation The greater the gradient the greater the Q 1 interval can be expected to be The gradient was measured from records similar to that in Figure 286*B* however the patient had atrial fibrillation (Courtesy of Braunwald *et al* (169) and *Circulation*)

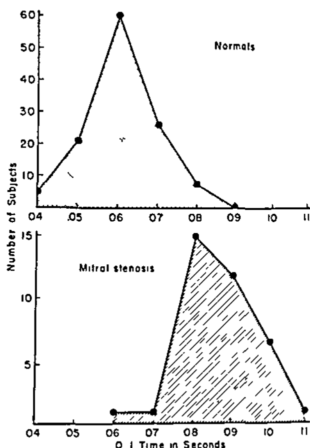


FIG 288 Q 1 time in patients with mitral stenosis compared with that in normal subjects (Courtesy of Craige (317) and the *New England Journal of Medicine*)

on the floor and one bounce the rumble corresponds to the sound of the ball rolling down the alley the snapping first sound is the impact of the ball on the pins

An *early systolic click* generated in the dilated *pulmonary artery is frequent in mitral stenosis* It is loudest in the pulmonary area and may introduce a short murmur This sound (p 129) may be produced by snapping of the arterial wall under the impact of ventricular ejection The click usually occurs about 0 09 sec after the beginning of the QRS of the electrocardiogram The click occurs with dilation of the pulmonary artery due to other causes In the oscillogram and to a much lesser extent to the ear the systolic click can be misinterpreted as a delayed snapping first heart sound For example, in a patient with primary pulmonary hypertension or multiple emboli the radiologic findings may suggest mitral stenosis and the early systolic click may be taken for a snapping delayed mitral first sound

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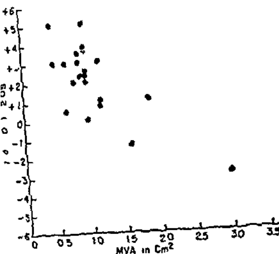


Fig 290 Relation of *Wells index* (Q 1 minus 2 OS) to the mitral valve area (MVA) as determined at operation or at cardiac catheterization by Gorlin s formula (Courtesy of Craige (317) and the *New England Journal of Medicine*)

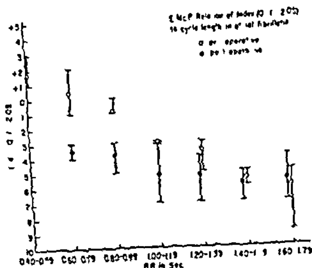


Fig 292 Relation of *Wells index* to cycle length in a patient with mitral stenosis and atrial fibrillation before and after mitral valve surgery

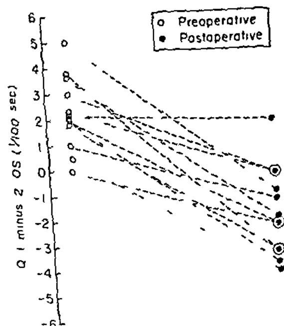
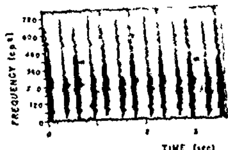


Fig 291 Wells index in 19 patients before and after valvulotomy for mitral stenosis (Courtesy of Craige (31) and the *New England Journal of Medicine*)



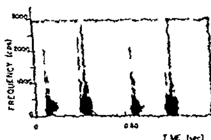


Fig 293 (*Above*) Pulmonary area in patient with high grade *mitral stenosis* There is a faint Graham Steell murmur (GS) (*Below*) Same area The record illustrates how it is possible to spread out the time base but with the original spectrographic method only at the expense of frequency span In this instance the heart sounds are sufficiently intense that especially with additional amplification two components are seen in the first sound and three in the second It is uncertain whether the components of the first sound are tricuspid and mitral closure sound or only one of these sounds plus an early injection snap There is a short early systolic murmur The three components in the region of S_2 are thought to be aortic closure sound pulmonary closure sound and mitral opening snap in this order

the diastolic murmur is the generally accepted near pathognomonic indication of mitral stenosis t the most worthy basis for such a ht

* Mitral regurgitation is included in the li t since given a certain minor degree of mitral obstruc

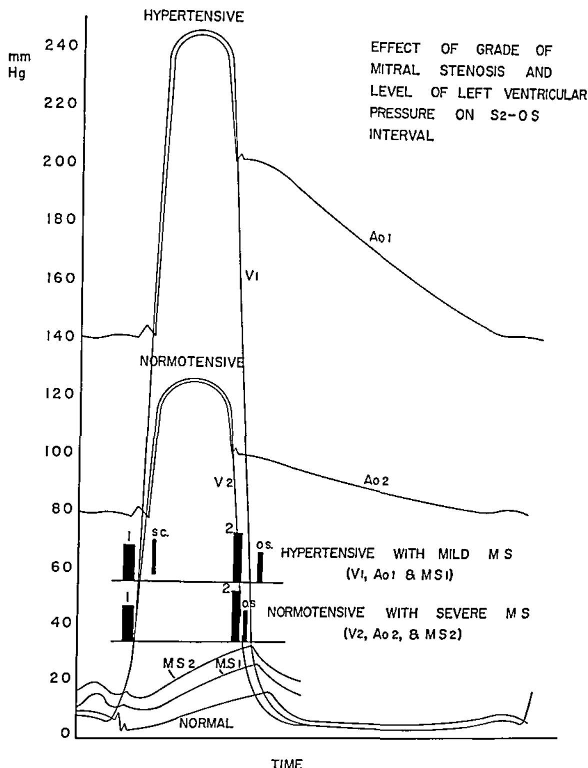


Fig 289 B

The Auscultatory Simulation of Mitral Stenosis

All that rumbles is not mitral stenosis
Hurst (734)

The list in Table 12 is a partial enumeration of the conditions which produce auscultatory signs similar to the diastolic murmur of mitral stenosis. The list could be extended by mention of conditions in which a snappy M_1 may be heard (p 286) in which pulmonary early systolic click and accentuated P_2 occur (p 289) in which the S_2-OS combination is imitated (p 290) and so on. But

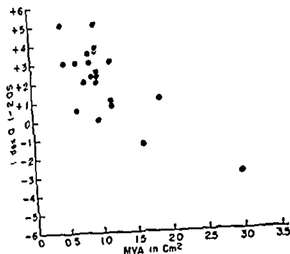


Fig 290 Relation of Wells index (Q 1 minus 2 OS) to the mitral valve area (MVA) as determined at operation or at cardiac catheterization by Gorlin's formula (Courtesy of Craige (31) and the New England Journal of Medicine)

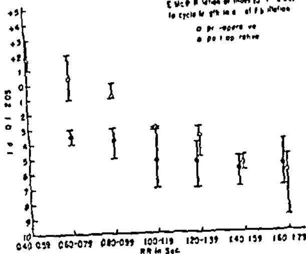


Fig 292 Relation of Wells index to cycle length in a patient with mitral stenosis and atrial fibrillation before and after mitral valve surgery

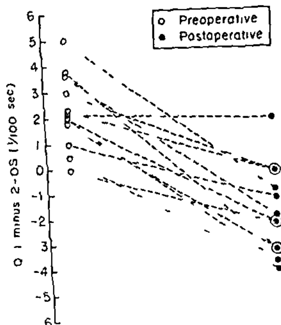


Fig 291 Wells index in 12 patients before and after valvulotomy for mitral stenosis (Courtesy of Craige (317) and the New England Journal of Medicine)

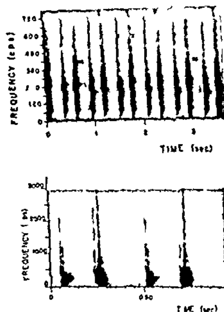


Fig 293 (Above) Pulmonary area in patient with high grade mitral stenosis There is a faint Graham Steell murmur (GS) (Below) Same area The record illustrates how it is possible to spread out the time base but with the original spectographic method only at the expense of frequency span In this instance the heart sounds are sufficiently intense that especially with additional amplification two components are seen in the first sound and three in the second It is uncertain whether the components of the first sound are tricuspid and mitral closure sounds or only one of these sounds plus an early injection snap There is a short early systolic murmur The three components in the region of S_2 are thought to be aortic closure sound pulmonary closure sound and mitral opening snap in this order

the diastolic murmur as the generally accepted near pathognomonic indication of mitral stenosis is the most worthy basis for such a list

Mitral regurgitation is included in the list since given a certain minor degree of mitral obstruc

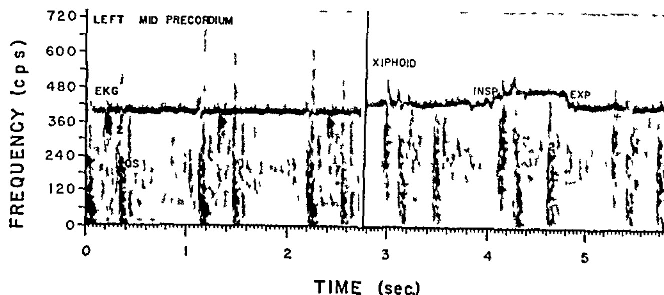


Fig 294 Fig 295

Figs 294 and 295 Systolic squeak of pericardial origin and atrial pericardial friction early after mitral valvulotomy

B C (305556) 35 years old demonstrates (in these recordings made one week after mitral valvulotomy) a late systolic squeak over the left midprecordium and a presystolic (atrial) friction sound over the xiphoid The S_2 OS interval is prolonged

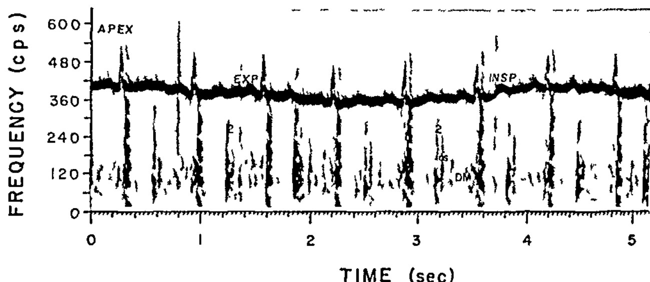


Fig 296 Effects of commissurotomy

The typical findings of mitral stenosis present before operation in E G (754164) are shown here After operation (the recording is *not* shown) S_1 was less delayed and the S_2 OS interval was increased An atrial friction rub was now present

tion the presence of mitral regurgitation will exaggerate the murmur which would be heard if no regurgitation were present The reason is that regurgitation produces increased mitral flow Not only must something approaching a normal quota of blood pass the valve but also that volume of blood regurgitated during the previous ventricular systole It is possible to imagine a degree of mitral stenosis which would produce no murmur were it not for the coincidence of mitral regurgitation

The Carey Coombs murmur of early rheumatic mitral valvulitis does not indicate permanent irreversible valve damage It is usually introduced by a third heart sound and is a blubbering mid diastolic murmur which *usually has no pre* systolic accentuation despite the presence of sinus

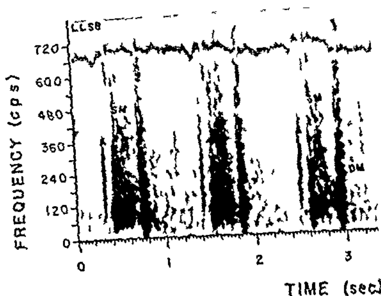


Fig. ?? LLSB in M P R (350?) six months after mitral commissurotomy for MS. S₁ told was said to be ... clear before operation. Note (1) relatively dull S₁ (described as snapping before operation) (2) decrescendo systolic murmur (3) a broad second sound which probably is fixed split (4) what is probably a third heart sound initiating a short diastolic murmur—all indications of mitral regurgitation probably created at surgery. Clinically the patient has improved. Note (1) huge ? wave of PKC (polarity inverted) and (2) the striking presystolic gallop which looks and sounded like a presystolic snap. The latter is a fairly frequent postoperative finding.

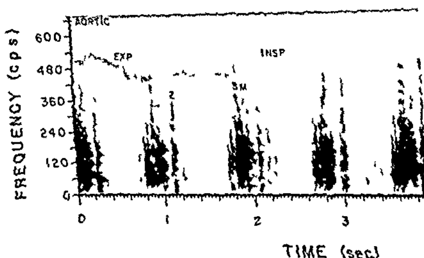


Fig. ?98 Exaggeration of murmur of AS after surgical correction of MS.
In D C (680606) the systolic murmur in the aortic area was not impressive before operation but provided unmistakable evidence of AS after operation as shown here.

rhythm. The reversible nature of the murmur is consistent with the view that it is due to edema in the valve plus dilatation of the ventricle. The latter is perhaps not essential since the murmur may occur in a heart which does not seem enlarged. Lavin and Darley (1248) point out with apparent validity that diastolic filling of the ventricle is more likely to be accompanied by sound in children witness the physiologic S₃. From the standpoint of auscultatory characteristic, the Carey Coombs murmur is the prototype of the murmur of relative mitral stenosis ... of

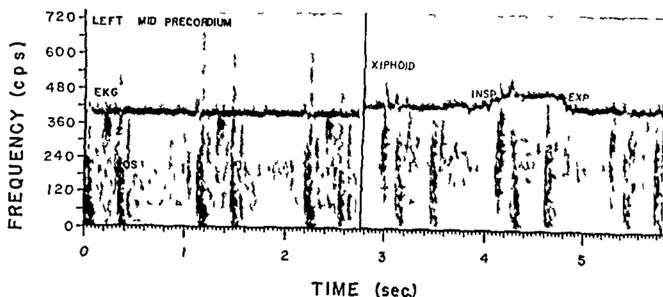


Fig. 294 Fig. 295

Figs. 294 and 295 Systolic squeak of pericardial origin and atrial pericardial friction early after mitral valvulotomy.

B. C. (305556) 35 years old demonstrates (in these recordings made one week after mitral valvulotomy) a late systolic squeak over the left midprecordium and a presystolic (atrial) friction sound over the xiphoid. The S_2 OS interval is prolonged.

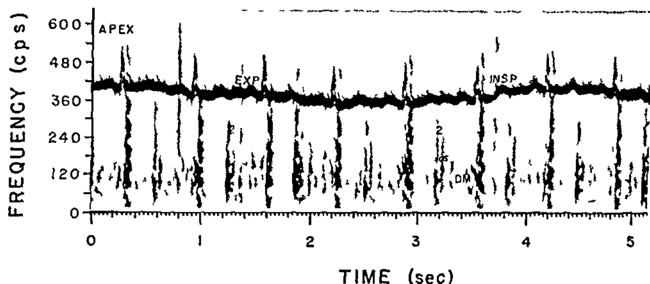


Fig. 296 Effects of commissurotomy.

The typical findings of mitral stenosis present before operation in F. C. (754164) are shown here. After operation (the recording is *not* shown) S_1 was less delayed and the S_2 OS interval was increased. An atrial friction rub was *now present*.

tion the presence of mitral regurgitation will exaggerate the murmur which would be heard if no regurgitation were present. The reason is that regurgitation produces increased mitral flow. Not only must something approaching a normal quota of blood pass the valve, but also that volume of blood regurgitated during the previous ventricular systole. It is possible to imagine a degree of mitral stenosis which would produce no murmur were it not for the coincidence of mitral regurgitation.

The Carey Coombs murmur of early rheumatic mitral valvulitis does not indicate permanent irreversible valve damage. It is usually introduced by a third heart sound and is a blubbering mid diastolic murmur which usually has no presystolic accentuation despite the presence of sinus

murmur In 2 1 AV di ociation po ibly in elderly persons in particular a Carey Coombs murmur may occur in early dia tole of the heart rate i proper A combination of rapid ventricular inflow and atrial s tole may be re pon ible With the econd P wave (which was followed by a ventricular contraction) there may be no murmur

Anemia particularly chronic anemia uch as that of hook worm disease and of ickle cell dis e e i a notorious mimic of mitral steno i (See the more detailed di cus ion on pp 417 to 419)

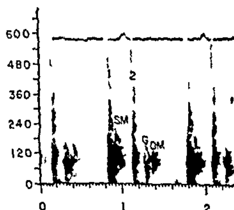


Fig 299 *Carey Coombs murmur at apex after exerci e* in 14 year old patient with acute rheumatic fever (I F A 5028) During a course of corti one in high dosage the gallop and rumble became *fixed even at re t*

M_1 is likely to be napping and there may be a pres tolic murmur or even a dia tolic murmur beginning in the earlier part of dia tole Dilata tion of the ventricle accelerated velocity of flow increased cardiac output and reduced vi co ity of blood are the factors operating in the complex causation of these phenomena

Figure 304 presents the crucial autop y finding in a patient thought clinically to have mitral steno si and regurgitation on a rheumatic ba i Autop y revealed syphilitic aortitis with a ingular coop deformity of the posterior aortic cu p such that regurgitation of blood again t the aortic leaflet of the mitral valve was likely to have occurred Di placement of the aortic leaflet by the regurgitant tream from the aorta has been a *favorite po i* bility for the mechani m of the Au tin Flint murmur Rarely doe one get such convincing evi dence as in this ca e Coul y (81) and Edward *and Burchell* (112) pre ented an autop y peci men in which a jet lesion developed on the aortic leaflet of the mitral valve clearly due to the stream of blood regurgitating again t it through the aortic valve (See page 283 for further discu ion of the Flint murmur)

In con trictive pericarditi I have observed a Carey Coombs type *of murmur* in two patient after operation (Fig 432) I have *no clear idea of*

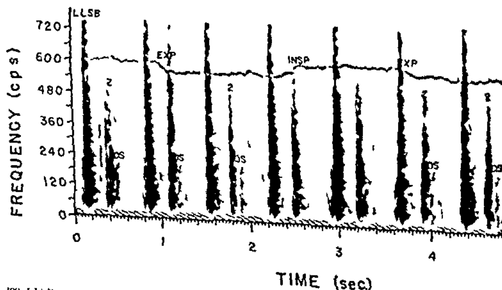


Fig 300 LLSB preop ratively in I B (443723) a year old female with *myx ma of left atri um* which was uccess fully rem ved urgically Sugge tion of a pre ystolic murmur and of an *opening nap* are seen Both the fir t and ec nd ound are accentuated and S is m*oderately d layed in relati on to the QRS*

TABLE 12

*Conditions with auscultatory signs simulating the diastolic murmur of mitral stenosis**

- I Conditions producing diastolic rumble at the apex
  - A Mitral regurgitation (see text)
  - B Carey Coombs murmur of early rheumatic mitral valvulitis
  - C High mitral flow (with dilated left ventricle)
    - 1 In congenital heart disease with left to right shunt
      - a Ventricular septal defect
      - b Patent ductus arteriosus
      - c Pulmonary arteriovenous fistula
    - 2 In complete heart block
    - 3 In anemia (1198)
    - 4 ? In thyrotoxicosis
  - D Tricuspid stenosis
    - 1 Rheumatic
    - 2 Relative
      - a In atrial septal defect or anomalous pulmonary venous return
      - b With pulmonary hypertension as in mediastinal collagenosis (J B 664434) multiple pulmonary emboli (D D 745792) etc (336 1263 1279 1595)
  - E Austin Flint murmur accompanying aortic regurgitation (p 283)
  - F Constrictive pericarditis
  - G Myxoma of the left atrium
  - H Malformation of left ventricular myocardium (336)
  - I Mere dilatation of the left ventricle as in congestive heart failure of any cause (1287 1520) probably especially if flow is relatively well maintained, as in so called high output failure
  - J Coarctation of the aorta (p 396)
  - K Congenital aortic stenosis (p 389)
- II Conditions mistaken for diastolic murmur
  - A Presystolic gallop
  - B Crescendic construction of first heart sound (107 738)
  - C Protodiastolic gallop

* See references 154 345 986 992 1520

TABLE 13

Causes of mitral regurgitation

- A Affecting the cusps predominantly
  - I Anatomic
    - 1 Rheumatic
    - 2 Bacterial endocarditis
    - 3 Congenital
    - 4 Traumatic
  - II Functional
    - 1 Atrial fibrillation
    - 2 Myxoma or ball valve thrombus (p 219 and 220)
- B Predominantly affecting the valve seat and suspension (i e the chordae and/or fibrous and muscular annulus)
  - I Ruptured papillary muscle or chordae tendineae
  - II Calcified annulus fibrosus mitralis
  - III Dilated ventricle on the basis of myocardial disease or the strain of systemic arterial hypertension or disease of the aortic valve

most of the examples in I of table 12 For example at times the term Carey Coombs is applied to the mitral murmur of ventricular septal defect (1590) The occurrence of this murmur which is sometimes very striking in association with congenital malformations (and the murmur of relative tricuspid stenosis with atrial septal defect) often raises the question as to whether congenital mitral stenosis is also present In atrial septal defect[5] because of dilatation the right ventricle constitutes the apex, and the murmur of relative tricuspid stenosis is heard there Even rheumatic stenosis of the tricuspid valve may be accompanied by a murmur heard fairly well out toward the apex In one case (I T 315167) of large pulmonary arteriovenous fistula (1393) there was a Carey Coombs murmur in the mitral area In location and quality it was quite distinct from the typical continuous murmur of the fistula itself

In complete heart block, Wood (1590) states that a diastolic murmur may be heard In over three fourths of the patients with congenital heart block of complete degree a functional mitral diastolic due to the large mitral stroke blood flow was heard particularly when the rate was under 50 Among 22 cases of congenital complete heart block referred to by Nadas (1137) half had an early or mid diastolic rumble Rytand (1335) described an atriodiastolic murmur in complete heart block in elderly persons The ventricle is necessarily dilated in complete heart block to accommodate the increased stroke volume Furthermore although the flow is spread over a longer period of time diastolic mitral flow is increased in volume and probably is particularly rapid during the ventricular filling phase of early diastole For these two reasons the occurrence of a functional diastolic murmur would not be surprising See p 442 for a discussion of the mechanism of Rytand's atriodiastolic

[5] See p 349 for a discussion of the numerous other respects in which ASD may simulate MS by auscultation

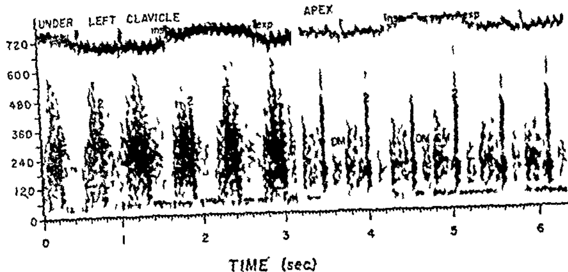


FIG 303 Recorded under left clavicle and at apex in B. A. (A93 18) with *patent ductus arteriosus proximal to coarctation*. In the former there is a typical continuous murmur with peak in late systole. In the latter there is a mid-diastolic rumble due probably to *torrential mitral flow*. The combination of coarctation with PDA exaggerates the left to right shunt. Associated fibroelastosis with mitral valve involvement is possible but not necessary to account for the murmur. S at the apex (aortic closure sound) is ringing. Hypertension may be the sole explanation but associated aortic valve disease (frequent with coarctation) is to be suspected.

presystolic when the patient is upright and systolic when supine. The first finding, rather pathognomonic of left atrial tumor or ball valve thrombus, was described in 1894 by [illegible] (1193) and in 1917 by Méroz (1015). In his monumental work on tumors of the heart, Mahaim (1023) referred to this phenomenon as "paradoxical mitral stenosis," since the murmur of ordinary mitral stenosis tends to disappear when the patient is upright.

Davis and Andrus (336) described one patient with what they termed "mitral stenosis in the male" produced by a congenital malformation of the myocardium of the left ventricle and a second patient with mediastinal collagenosis. In both M_1 and I were increased and there was a mid-diastolic rumble at the apex. In the second the diastolic murmur may have been of tricuspid origin (see p 328). One case described by Teare (1403A) under the title "Asymmetrical hypertrophy of the heart in young adults" seems to have been the same condition as in the first case of Davis and Andrus. Teare's case was also subjected to operation for presumed mitral stenosis.

Occasionally a diastolic rumble is present during heart failure and disappears with restoration of compensation (461). I observed this sequence of events in a 76-year-old man with congestive failure and atrial fibrillation on the basis of coronary arteriosclerosis. Usually the sequence of events in true mitral stenosis is the converse: the diastolic murmur is likely to become inaudible during failure. Mere dilatation of the ventricle without failure necessarily may result in a diastolic rumble suggesting mitral stenosis, as in a 27-year-old individual with coronary artery disease who was subjected to mitral valvulotomy (see Fig 453 p 422). Although obviously not unique to Friedreich's ataxia, one can point to the mid-diastolic and presystolic murmur which has been described (312, 662, 1212, 1379, 1180) in association with the myocardiopathy of this primarily neurologic disorder as another example of the simulation of mitral stenosis by myocardial disease. Atrial dysrhythmias also occur fairly frequently in these cases, increasing the simulation of rheumatic heart disease in these young individuals in whom the involuntary movements may be thought to represent Sydenham's chorea and aching of the legs occur commonly.

In the early part of the first heart sound there is often a low pitched component formerly

the mechanism, although dilatation of one or both ventricles was probably present in both and the first approximation to an explanation would appear to be dilatation of the ventricle, resulting from partial disuse atrophy and some fibrosis, in association with AV rings which are normal in dimension and possibly are kept normal by scar tissue in the AV groove Also without operation in constrictive pericarditis a mid diastolic murmur has been described (263, 461)

With one possible exception, myxoma of the left atrium (and ball valve thrombus (1603), which usually occurs only when there is mitral valve disease with an element of stenosis) can produce all the auscultatory signs of mitral stenosis—delayed, snapping, M_1, diastolic rumble at the apex accentuated P The possible exception is the mitral opening snap (1023) However, a diminutive sound of this type may be demonstrable (see Fig 302) In a case of myxoma with phonocardiograms of presumed opening snap recently published (868), the interval between S and the extra sound is consistent with the interpretation offered However, the phonocardiogram demonstrates a longer sound than the opening snap usually is Possibly it introduced a short rumble In another report (261) it is stated that "an opening snap was evident" stethoscopically The mitral valve was normal at autopsy In his patient Ludwig (970) described the presence of *Wachtelschlag* sound of the quail Since this was a simile used by German writers (1214) for the second sound and opening snap following in close succession it has been assumed by subsequent authors (1023) that an opening snap was present in that case As to the murmur of myxoma some (116 824 1023) have claimed it is diastolic and

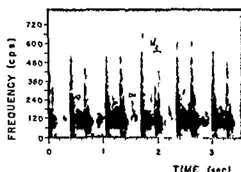


Fig 301 Mid diastolic rumble at apex with atrial septal defect (M M A76511) Split S_2 also characteristic of ASD

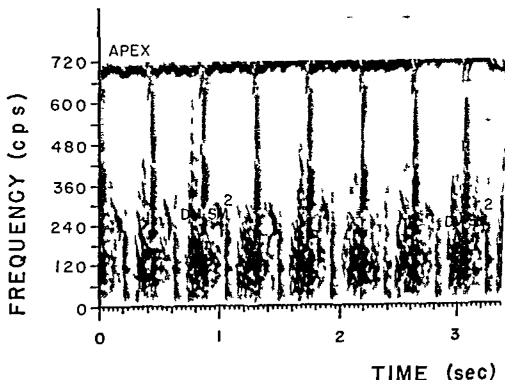


Fig 302 Apex in D T (B6445) with AV communis Note diastolic murmur which because of the rapid rate is a summation of passive and active murmurs Evidences of slight musicality of the systolic murmur (see Fig 187)





Fig. 50. Traumatic mitral regurgitation.

Adams' case (6) of a 20 year old man who died in Wenckebach's clinic in Vienna in 1924, ten years after attempting suicide with a revolver. The bullet entered in the fourth interspace two fingersbreadth below the nipple. He was seen in the Surgical Clinic and operated on. [illegible] Left pneumothorax and sizable hemopericardium were found; the bullet, which by x ray was seen to move with the left ventricle, was not recovered.

Thereafter the patient showed a grating apical systolic murmur and also a loud systolic murmur in the pulmonary area. Relative tricuspid insufficiency, atrial fibrillation and congestive failure developed.

The sections shown here were made after allowing the thoracic organs to fix for 48 hours in formol. (Above) *Posterior portion of the first cut.* The oval hole in the anterior cusp of the mitral valve measured 18 by 14 mm. Both atria (the *septum* and part of the right atrium with entering venae cavae are seen) were tremendously dilated, *compressing the right lung.* In fact the heart extends practically from right axilla to left axilla. The dilated hepatic veins and the *nutmeg liver* of chronic passive congestion are seen.

(Below) *Anterior portion of the second cut.* The *left* ventricle is seen in this view as in the prior one. The interatrial septum and fossa ovalis are visualized. The bullet was found *embedded* in scar tissue in the region of the apex of the left *ventricle*. Again the tremendous atrial dilatation is evident.

Rheumatic damage affects most commonly the mitral valve (see p. 261); mild mitral regurgitation is the most frequently occurring sequel of rheumatic fever. It is this type of mild lesion furthermore which, perhaps only for reasons of its high frequency, is most often affected in subacute bacterial endocarditis. Pure mitral regurgitation, unlike pure mitral stenosis, is more frequent in men.

Congenital mitral insufficiency occasionally is found alone but usually is associated with another malformation, most often the ostium primum variety of atrial septal defect.

Traumatic mitral regurgitation is indeed rare. An instructive and remarkable case is that of a Viennese man (6) who in 1914 attempted suicide with a pistol and survived until 1924, dying of the effects of pure mitral regurgitation from a clean hole in the anterior (aortic) leaflet of the mitral valve (see Fig. 50).

It is because the valve cusps and chordae tendineae are so important to the competence of the AV valves that mitral regurgitation may occur with a great variety of conditions which in some way or ways affect the noncuspal portion of the valve mechanism.

Following myocardial infarction, a papillary muscle may rupture with resulting mitral regurgitation. Mere infarction and scarring of the papillary muscle can also result in mitral regurgitation through failure of the mechanism by which the

thought to be of atrial origin (1166, 1244)—and indeed they may be in many cases, as discussed on p. 126)—but considered to arise sometimes from contraction of the ventricular myocardium, since it was demonstrated (308) that it may persist in atrial fibrillation. When particularly striking, the low pitched "mitral vibrations" followed by a sharp valve closure sound can suggest a presystolic murmur and accentuated M_1 of mitral stenosis (536). Bramwell and Ellis (157) made the interesting auscultatory observation, which unfortunately did not have graphic documentation and analysis, that in 12 of 192 athletes there was a "curious prolongation of the first heart sound, not unlike the crescendo murmur of mitral stenosis." Three were Marathon runners, three were long distance runners and three were cyclists. The phenomenon was not present in any of 18 sprinters or 16 middle distance runners. In 1909 Sewall (1378) described "first sounds beginning with a crescendo tone, simulating closely the faint and brief presystolic murmur."

MITRAL REGURGITATION

(Syn. Mitral insufficiency, mitral incompetence, MR)

ETIOLOGIC AND ANATOMIC CONSIDERATIONS (194). Although the etiologic possibilities for true mitral stenosis are really only two (with many simulating conditions), the causes of mitral regurgitation are numerous (see Table 13, p. 306). Relatively the same situation exists in connection with aortic stenosis and aortic regurgitation (see p. 269).



FIG. 304. S. A. S. (269149). A 44 year old female had had congestive heart failure for two months and murmurs interpreted as those of mitral stenosis and regurgitation, presumably rheumatic. At autopsy there were no stigmata of rheumatic fever but the aorta showed florid changes of syphilitic aortitis. There was a "sugar scoop" deformity of the posterior cusp of the aortic valve so positioned as to direct blood against the aortic leaflet of the mitral valve which shows a jet lesion just below the prolapsed cusp of the aortic valve.

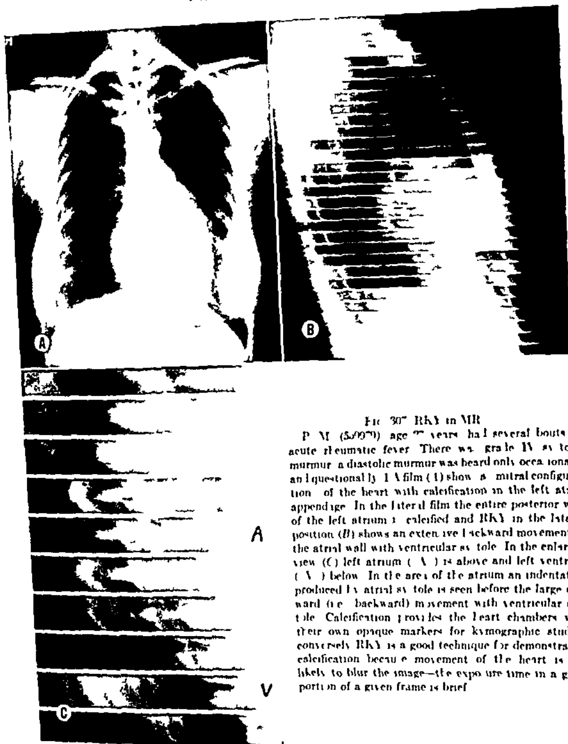


FIG. 307. RKY in MR

P. M. (5,0070), age ?? years, had several bouts of acute rheumatic fever. There was grade IV systolic murmur; a diastolic murmur was heard only occasionally and questionably. A film (A) shows a mitral configuration of the heart with calcification in the left atrial appendage. In the lateral film the entire posterior wall of the left atrium is calcified and RKY in the lateral position (B) shows an extensive backward movement of the atrial wall with ventricular systole. In the enlarged view (C) left atrium (A) is above and left ventricle (V) below. In the area of the atrium an indentation produced by atrial systole is seen before the large outward (i.e., backward) movement with ventricular systole. Calcification provides the heart chambers with their own opaque markers for kymographic studies; conversely, RKY is a good technique for demonstrating calcification because movement of the heart is less likely to blur the image—the exposure time in a given portion of a given frame is brief.

hot gun or more specific fashion for febrile illness, healed bacterial endocarditis is likely to become a more frequent primary cause of regurgitation at the heart valves or important exaggerating factor in deformities already present.

Hepper and colleagues (674) describe a 79 year old woman in whom a harsh systolic murmur could be accounted for on the basis of healed and calcified bacterial lesions of the mitral valve.

In autopsies on 177 individuals 40 years of age



FIG 306 Calcified annulus fibrosus mitralis in 88 year old woman (M S 481084) with rheumatic fever in childhood (*A*) Exposure time 1/60 sec (*B* and *C*) RKY In the enlarged view (*C*) it is seen that the calcified zone and the ventricular border move toward each other in ventricular systole The movement of the base of the heart as labelled by the calcified mitral ring is of large amplitude Descent of the base of the heart in ventricular systole was demonstrated by Leonardo da Vinci (774) Percutaneously he introduced three needles in the heart of a living animal one at the apex one at the base one in the middle With each ventricular systole the middle needle moved little whereas the external ends of the apical and basilar needles moved away from each other Systolic descent of the base has been demonstrated by others (1453) in patients with calcified mitral annulus and in animals in which radio paque markers were placed in the heart (1531) An unusually loud harsh holosystolic murmur was present in this patient A protodiastolic gallop characteristic of mitral regurgitation was also demonstrated

papillary muscle shortens the effective length of its attached chordae during ventricular systole

Mitral regurgitation occurs with calcification of the annulus fibrosus mitralis (Fig 306) a condition which is poorly understood from the standpoint of pathogenesis, but which occurs in older people (1337) and sometimes in conditions favoring metastatic calcification, such as multiple myeloma Rheumatism sometimes seems to be involved in its causation Complete heart block is sometimes associated The rigidity of the fibrous skeleton apparently interferes with contraction of the muscular ring which ordinarily serves a significant role in narrowing the AV orifice and assisting competent AV valve closure It is interesting that the tricuspid ring is so rarely involved relative to the mitral ring

With the widespread use of antibiotics in

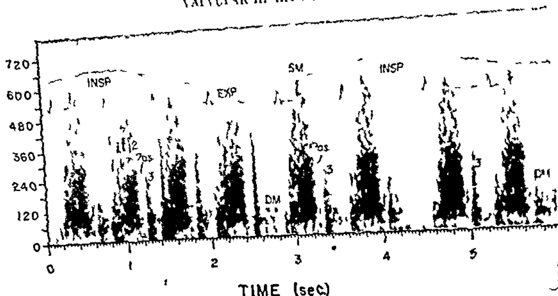


FIG 310 Rheumatic mitral regurgitation

In this case the systolic murmur begins with the second component of the first sound (mitral closure). The first sound is dull. In this case the systolic murmur although holosystolic is crescendo rather than *decrescendo*. A striking third heart sound gallop is present. This shows variation with respiratory phase being more conspicuous during expiration. It is followed by a short low pitched murmur (*rumble*). In the gap between the second snap and the third sound there is a faint sound. That this is an opening snap and not the second part of a split second sound is suggested by the fact that its relationship to the second sound *shows* no variation with respiration. The presence of an *opening snap* in record as recorded as this for demonstrating it is not necessarily evidence that significant mitral stenosis is present. On the other hand its absence would be strong evidence against the existence of significant mitral stenosis.

motion of any sort. Douglas (367) points out that occasional cases of predominant mitral regurgitation may result from scarring and shortening of the chordae tendineae which hold the cusps open during ventricular systole. In such cases the cuspal curtain may be *mobile and* M_1 paradoxically accentuated.

The systolic murmur of mitral regurgitation is thought to be holosystolic in the great majority of cases. The rise and fall of *interventricular* pressure are rapid with most of systole spent at or near peak pressure—a reason to expect a holosystolic murmur with mitral *regurgitation*. Vaquez quoted by Castex (215) thought dilatation of the heart might result in a non holosystolic early or late systolic murmur. This is questionable however. As pointed out on page 289 an early decrescendo non holosystolic murmur *occurs* rather commonly in cases of mitral stenosis. Although the mechanism is not entirely *clear the* murmur probably *is produced* by mitral regurgitation. It *may* be that left ventricular *pressure* early in systole is sufficiently higher than the pressure in the recently although only partially decompressed left atrium to produce mitral regurgitation but that as left atrial inflow continues and after the effects of descent of the base of the atrium are cancelled out there is inadequate pressure *gradient* to produce continued regurgitation and murmur.

The holosystolic murmur may be plateau decrescendo or crescendo. The anatomic hemodynamic basis *for* these three different patterns is not clear. Particularly it is hard to understand the basis for a murmur which has its greatest *intensity late in systole*. A plateau murmur is explicable thus *is the usual* pattern with high *grade mitral* regurgitation. A decrescendo pattern *probably* results from *the fact that* although pressure in the left ventricle is relatively *fixed* during ventricular *systole* pressure in the *atrium* increases during the course of systole.

The systolic murmur is usually loudest at the cardiac apex and *is well transmitted* to the left axilla. With mammoth left atrium especially in young patients with small chests it is well heard

Movitt and Gerstl (1126A) questioned the benignity of pure mitral regurgitation when they reported autopsy studies of four men who died of heart failure from this lesion at the age of 42, 50, 51 and 56 years. Olesen and Warburg (1153) followed up 14 persons under 30 years of age in whom a *loud* apical systolic murmur was the only finding. The average period of follow-up was 11.8 years. Three of the patients had died.

Since the development of mitral valvulotomy for mitral stenosis, mitral regurgitation has taken on a more serious aspect. The association of mitral *regurgitation in significant proportions* usually has the effect that the benefit of surgery for the mitral stenosis is less than maximal. In addition, it is now appreciated that there is a small minority of cases of pure mitral regurgitation of such severity that it is a major source of embarrassment to the circulation (see above).

In mitral regurgitation in contradistinction to aortic regurgitation, the regurgitant volume may exceed the forward flow volume without there being any elevation of end-diastolic pressure in the ventricle.

Pressure curves from the left atrium in both experimental and clinical mitral regurgitation *(recorded at operation or by transbronchial* puncture or direct puncture from the back) and volume curves recorded with balloons in the esophagus demonstrate a late systolic peak. This may be related to the crescendo type of holosystolic murmur which some patients with MR display.

*Several colligative phenomena in mitral re*gurgitation can be listed: the protodiastolic gallop, rapid outward movement (827) of the left ventricular wall in early filling (by electrokymography), rapid descent (steep slope) on the Y limb of the left atrial pressure curve (1172), i.e., that part corresponding to early ventricular filling, rapid inward movement (760) of the left *atrial border in the phase corresponding to early* ventricular filling (by EKY), the impact on the chest wall which Harvey (652) called "ventricular knock." If the last is to be distinguished from the protodiastolic gallop it should probably be applied to the mechanical phenomenon readily appreciated by palpation.

Any valvular regurgitation represents in essence an internal shunt. Therefore, certain parallels with the hemodynamics and auscultatory findings of the shunts of congenital septal defects are to be expected. For example, mitral regurgitation and ventricular septal defect have points of hemodynamic and clinical (v. infra) similarity.

CARDIOVASCULAR SOUND. *The auscultatory fea*tures of mitral regurgitation (Fig. 309) are (1) dull first heart sound at the apex, (2) holosystolic apical murmur, (3) splitting of the second sound at the base, (4) exaggerated third heart sound which may be followed by (5) a short mid-diastolic rumble.

Apparently the presence of appreciable mitral leak prevents the sharp tensing of the aortic leaflet of the mitral valve necessary for the production of a *first heart sound* of normal intensity and snappiness. The first sound may be virtually absent in MR or have only components *of low* frequency and intensity which endow it with the "dull" quality as appreciated by the stethoscopist. Often the valve in cases of predominant regurgitation is heavily calcified and incapable of much

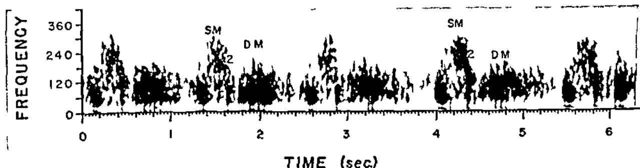


Fig. 309 Predominant *mitral regurgitation* in L. L. (227489) apex. Note (1) low frequency content of dull S_1, (2) relatively high pitch and crescendo character of the systolic murmur, (3) third heart sound initiating diastolic rumble, (4) absence of presystolic murmur because of atrial fibrillation.

relatively high frequency about 800 c p s The result is a murmur to which the analogy of the cooing dove is appropriately assigned

The murmur of ruptured papillary muscle or chordae tendineae (Fig 319) is usually described as loud rough harsh and coarse (44) Some refer to it as musical that this is not more often the case is probably because of differing views on what constitutes musicality Schwartz and Conolly (1332) reported the following auscultatory findings *in a case of ruptured papillary muscle* A loud harsh musical systolic murmur was *heard in the* mitral area and was transmitted into the left axilla and posteriorly to the region of the left *scapula The murmur filled the entire period of* systole and ended with the second heart sound

The systolic murmur of aortic stenosis is well *transmitted to the apex and may cause confusion The phonocardiogram permits differentiation* since the murmur of aortic stenosis has a characteristic shape and stops before the second sound (p 268) *The systolic murmur of tricuspid regurgitation* may be well heard or even *loudest* at the apex although transmission into the axilla is usually not striking In cases of pure *mitral stenosis tricuspid regurgitation* may create *a false* impression of associated mitral regurgitation

The second sound at the base is often split in mitral regurgitation (Fig 312) Early closure of the aortic valve results from the fact that the left ventricle has two portals of discharge It is less impressively exaggerated in mitral regurgitation than mitral stenosis because *pulmonary hypertension* is usually less marked

An exaggerated *third heart sound* is an important and constant sign of mitral regurgitation It is the result of increased left *ventricular filling* in early diastole the increase involving both volume and rate Often one feels a sharp *impact of the* ventricle against the chest wall at *the time of the* loud third heart sound a feature which led Harvey to suggest (1052) the designation of ventricular knock " Often the third sound is the loudest of the heart sounds (e g Fig 313) *One must be careful not to confuse* the loud third sound for the second heart sound This is easily done because *the second sound* is likely to be buried in the last of *the murmur*

The third sound may be followed by a short *diastolic rumble* even though there is little or no mitral obstruction Rapid and large mitral flow *and the dilatation of the left ventricle are responsible*

Frequently the spectral phonocardiogram demonstrates in cases of *predominant mitral re*

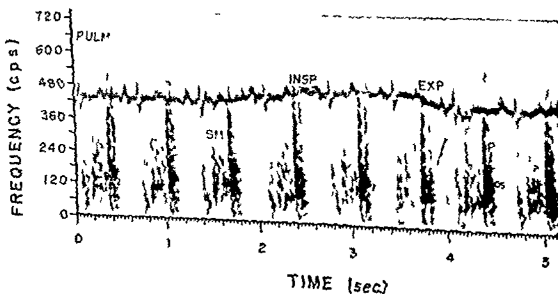


Fig 312 Split S with *mitral regurgitation*
B C 33 1 02 23) a 74 year old female also has an early diastolic *murmur* at the left sternal border The degree of splitting is exaggerated by inspiration

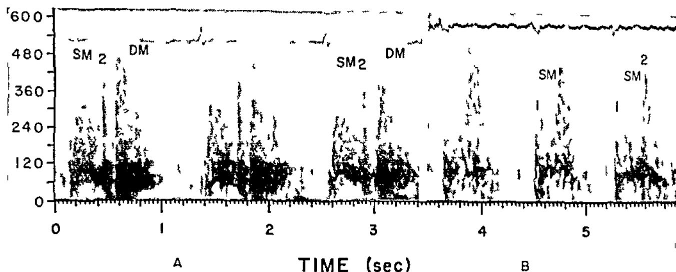


Fig 311 Rheumatic mitral regurgitation

The recording at left was made from a patient who was explored with the intent of performing mitral valvulotomy. Pronounced mitral regurgitation and very little mitral obstruction were discovered. At the apex the first sound is muffled. Possibly two components can be faintly discerned. A decrescendo systolic murmur begins immediately with the second component which is probably a delayed mitral closure sound. The second sound at the apex, which probably has its origin largely in closure of the aortic valve, is followed by a gap before the beginning of the diastolic rumble. The diastolic rumble begins abruptly with a third sound gallop. No opening snap is demonstrated. The protodiastolic gallop is exceedingly loud, louder in fact than the normal heart sounds.

(*At right*) the systolic murmur is crescendo in type. Right bundle branch block is present. Splitting of the second sound is barely discernible in some cycles. The murmur continues directly on into early diastole. It is not unexpected that in some anatomic varieties of regurgitant mitral valves regurgitation into the left atrium should continue after closure of the aortic valve and until the time that pressure in the left ventricle has fallen below that in the left atrium. Then a reversal of flow will occur. The net result is that a continuous to and fro murmur occurs; this is rare however.

posteriorly in the interscapular areas and up and down the back, often even over the sacrum (1478)[6] or occiput. Furthermore it may be audible to the right of the sternum in the same area in which an expansile systolic pulsation may be seen—about the third right interspace a centimeter or two from the costal margin.

Osmundsen *et al* (1170A) and Edwards and Burchell (412) described a case of mitral regurgitation from ruptured posterior chordae tendineae in which aortic stenosis was simulated because of a systolic murmur and thrill to the right of the sternum and even into the carotid arteries. At necropsy a jet lesion of the left atrial endocardium overlying the interatrial septum was found. Movitt and Gerstl (1126A) also had a case of pure mitral regurgitation with systolic murmur in the aortic area and carotids. In another case (274A) of ruptured posterior chordae tendineae a harsh grade IV systolic murmur was loudest at the apex but also loud in the aortic area and carotids. A misdiagnosis of aortic stenosis was made. The case is less definite because the aortic valve was calcified but not stenosed. Rytand and Lipsitch (1337) had a case of calcified annulus fibrosus mitralis with mitral regurgitation and a systolic murmur well heard in the aortic area and over the carotid arteries.

White (1537) thinks the murmur of mitral regurgitation is louder during heart failure and less intense when myocardial function improves. If the observation can be confirmed by objective measurement it will accord well with the finding of Wiggers and Feil (1557) on the relationship between the speed of ventricular contraction and the volume of regurgitation (see above).

The quality of the murmur may be blowing or musical but almost never harsh. A systolic thrill is sometimes felt. A musical murmur is particularly likely to be present during acutely active rheumatic carditis and during and after bacterial endocarditis. The musicality in such cases is usually represented by a single harmonic at

[6] See patients D R S (A 2/22) and G J (575841)

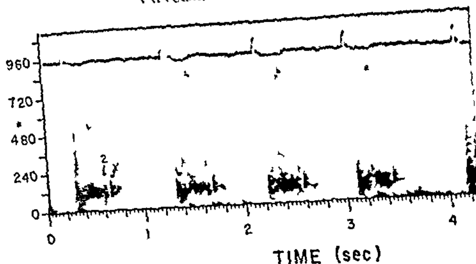


Fig 316 Musical systolic murmur at apex in 60 year old patient (I. B. 1 0 45) with superannuated rheumatic heart disease and *mitral regurgitation* (Note high frequency of the single harmonic)

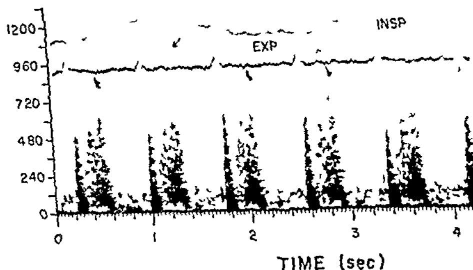


Fig 31[illegible] Apex in E. F. (1 14 15) with the Marfan syndrome and probable *mitral regurgitation* partly on the basis of ventricular dilatation partly on the basis of redundant, hereditarily weak chordae tendineae. *To the ear* delicately musical quality like wind blowing through naked limbs (see) (Note high pitch of single harmonic)

ejection in each case A holosystolic murmur and by graphic means a mild splitting of the second sound occur in both conditions In young people with small chests the murmur may be audible over a large area of the chest and the classical areas of maximum audibility may not be clear The presence of a loud third sound gallop may point to mitral regurgitation but is found with VSD as well (Fig 360)

After much discussion and testing of methods for identifying predominant mitral regurgitation some expensive arduous and perhaps hazardous to the patient many cardiologists have come to the conclusion that cardiovascular sound provides the surest and simplest indicator It may be *appropriate* to conclude with a quote from Arthur Ernest Sansom (1839-1907) of the London Hospital writing in his *Valvular Disease* of 1883

It is only comparatively recently that our pupils could be taught in our hospitals the methods of discriminating

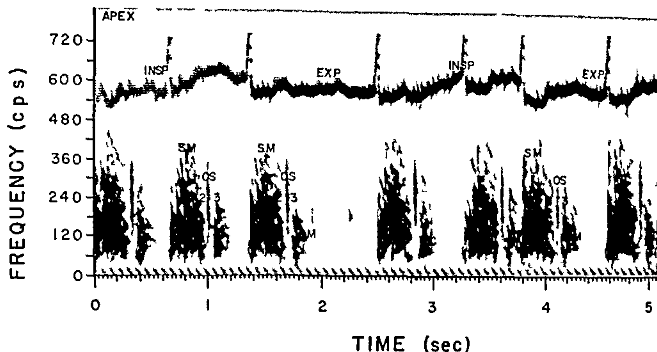


Fig 313 Predominant mitral regurgitation

Apex in M P (767600) 16 year old female with advanced rheumatic heart disease The clearly demonstrated opening snap does not contradict the diagnosis of predominant mitral regurgitation

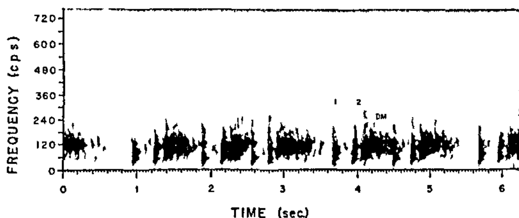


Fig 314 Mitral stenosis with considerable mitral regurgitation atrial fibrillation

gurgitation a diminutive opening snap between the second and third heart sounds (Fig 313)

Simon and Liu (1391) have emphasized the harsh or musical holosystolic apical murmur which may be associated with calcified mitral annulus Ashworth (34) found an apical systolic murmur in five of ten cases of calcified mitral annulus

More often than one might perhaps think there is a problem in the differential diagnosis of mitral regurgitation and ventricular septal defect of the Roger type There are hemodynamic similarities inasmuch as the left ventricle has two ports of

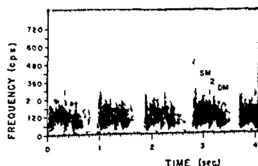


Fig 315 Predominant mitral regurgitation Essentially continuous murmur Opening snap situated in interval between S and S₃ the latter introduces the main part of the diastolic murmur

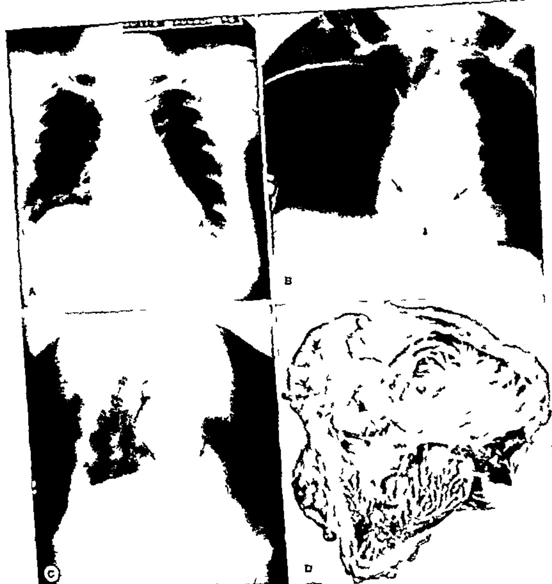


Fig. 320 Myxoma of the right atrium

B. J. (60(384 ant. 23 81) old year old woman had for six years had clinical features usually considered typical of constrictive pericarditis. Exploratory thoracotomy for constrictive pericarditis revealed no pericardial abnormality. In addition to hepatomegaly and ascites the patient displayed a somewhat inconstant diastolic rumbling mid diastolic murmur at the left border of the lower sternum. In the conventional film the superior vena cava is dilated and the right atrium bulges to the right. The angiocardiogram (*B* and *C*) show the dilated superior vena cava and in *C* the enlarged azygos vein. There is a large ball like filling defect in the right atrium. At autopsy the heart showed remnant of a large polypoid tumor in the right atrium (*D*). The greater portion of the tumor had been removed surgically 24 days before death. (Case of Bahn and Newman (42); illustrations courtesy of Cooley and Stein (201).)

areas such as the ovary which are drained directly into the vena cava. Myxoma (Fig. 320) (42) other ball valve neoplasm (Fig. 321) and ball valve thrombus (L.C. 646029) occur on the right side of the circulation as they do on the left with production of auscultatory signs similar to those of rheumatic tricuspid stenosis. Relative tricuspid stenosis with diastolic murmur may occur with high flow and dilated right ventricle in atrial septal defect and anomalous pulmonary venous return. In *rare instances* relative tricuspid stenosis occurs in case of

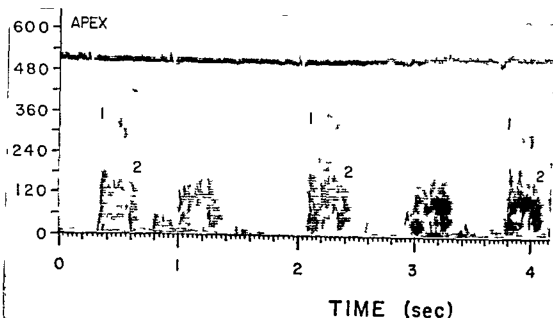


FIG 318 Musical apical systolic murmur in patient (A C 580081) with obscure myocardiopathy and dilated ventricle

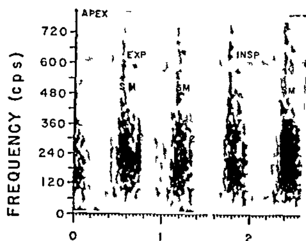


FIG 319 Ruptured chorda tendinea
Harsh loud systolic murmur of mitral regurgitation which developed in the last year of life in I D (690316) 73 years old There is a faint presystolic crescendo murmur Autopsy revealed a ruptured chorda tendinea histological clusters of bacteria (cocci) were demonstrated Other than the appearance of the murmur there were no clinical clues to the diagnosis In addition to the holosystolic murmur there is both a protodiastolic and a presystolic gallop (indistinctly shown here)

between cases of mitral stenosis and those of mitral regurgitation and it is unwise to conceal the fact that difficulties in such differential diagnosis do occur

I need not ask you to concur with me in deprecating the pleas of *Cui bono?* It is our bounden duty to learn all we can of the disorders we have to treat even if the immediate influence of such knowledge upon treatment be not so very apparent

TRICUSPID STENOSIS (TS)

ETIOLOGIC AND ANATOMIC CONSIDERATIONS (569 A 712, 1056 1057) Rheumatic fever is the most frequent cause of this relatively uncommon lesion Recently, a case of isolated tricuspid stenosis presumably rheumatic has been reported (572) Usually however, there is involvement of the mitral and aortic valves as well Gibson and Wood (548) never found tricuspid stenosis with severe mitral regurgitation They had one case in which lupus erythematosus seemed to be the cause Congenital tricuspid stenosis (p 382) is occasionally encountered Fibroelastosis can be the basis (1218)

Bailey and Bolton (43) found significant tricuspid stenosis in 15 of 98 patients with mitral stenosis in which the tricuspid valve also was explored It is not clear to what extent the incidence in this series was exaggerated by preoperative clinical suspicions of tricuspid stenosis dictating tricuspid exploration

Because the endocardium of the right atrium is normally thin and the right atrium more distensible than the left a paper thin papyraceous atrium may occur (1277)

Recently it has become known that by some mechanism scarring occurs in the valves on the right side of the heart when metastasized carcinoid tumors are present in the liver and other

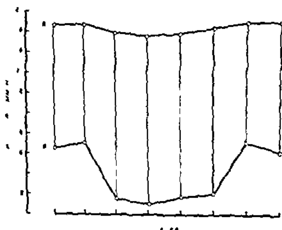


FIG 32.1 Variations in right atrial and right ventricular pressure (and by derivation in transtricuspid gradient) with respiration in patient with tricuspid stenosis The third to the sixth cycles occurred during inspiration During inspiration right ventricular pressure falls with the fall in intrapleural pressure any fall in right atrial pressure is almost completely offset by venous inflow (From Killip and Lukas (702A))

valve averaged 6 mm Hg in the 12 patients of Gibson and Wood (548) The end-diastolic pressure gradient is of most significance When there is sinus rhythm giant a waves in the jugular vein and presystolic pulsation of the liver may occur This is a nonspecific finding since any condition such as constrictive pericarditis with elevated end-diastolic pressure in the right ventricle has the right atrium contracting against resistance Giant a waves are seen in pure pulmonary stenosis and in primary pulmonary hypertension (q.v.) The presystolic gallop is probably a correlated phenomenon

CARDIOVASCULAR SOUNDS Tricuspid stenosis produces many of the same auscultatory signs as does mitral stenosis particularly an opening snap and a diastolic murmur with presystolic accentuation A discussion directed mainly at distinguishing features is in order We have not been impressed with an accentuation of the first heart

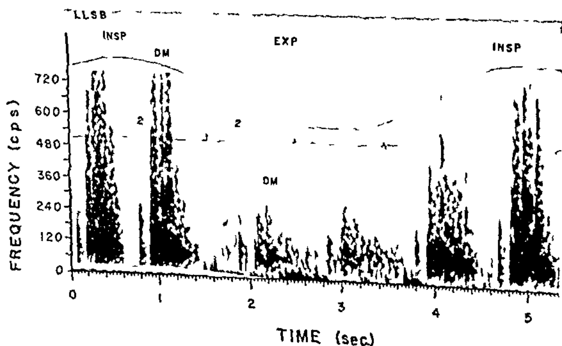


FIG 32B Tricuspid stenosis

LLSB in D. D. (5 8379) 30 year old *Atrial Fibrillation* Mitral valvulotomy performed 3 years previously Presistent hepatomegaly and mild hyperbilirubinemia bulging right atrium at the right heart border and a diastolic rumble with thrill at the left sternal border suggest tricuspid stenosis The signs seemed to become more striking after mitral valvulotomy possibly because of increased blood flow Note the marked accentuation of the murmur (in intensity and frequency span) and shortening of the S murmur interval with inspiration The second sound could be confused for first sound and the murmur thought to begin with a second heart sound One year after this recording the murmur was constant in intensity throughout the respiratory cycle After tricuspid valvulotomy the diastolic murmur again showed respiratory variation



Fig 321 Occlusive tumor of right atrium
R B (545329 aut 22591) 59 year old man had the clinical picture of constrictive pericarditis The carcinoma producing the ball valve tumor was of uncertain origin

pulmonary hypertension apparently on the basis of dilatation of the right ventricle

PHYSIOLOGIC CONSIDERATIONS (457 1600) The most important functional aspect from the point of view of cardiovascular sound is the effect of inspiration on the diastolic murmur The fall in intrathoracic pressure with inspiration increases venous return ("thoracic aspiration , respiratory pump") and in turn increases the diastolic murmur of tricuspid stenosis Killip and Lucas (792A) have demonstrated that the increased tricuspid gradient with inspiration is the result of drop in right ventricular pressure Pressure in the right ventricle follows intrathoracic pressure closely and drops during inspiration whereas any drop in right atrial pressure is cancelled by increased inflow of blood See Fig 322A

The diastolic gradient across the tricuspid

of the murmur in tricuspid stenosis evident in Figures 322 and 325E cannot be taken as an indication that the murmur would impress the ear as being higher pitched. Probably the greater intensity of the component at lower frequencies outweigh the high frequency components. However, Chesterman and Whitaker (258) state that the diastolic murmur may be high pitched and at times have a sea gull quality. This I have not observed.

So intense is the diastolic murmur that the unwary can easily misinterpret it as a systolic murmur. Although the diastolic murmur of tricuspid stenosis is usually loudest in the region of the lower left sternal border one must be prepared to find its point of maximal audibility at least as far to the left as the midclavicular line.

The murmur of relative tricuspid stenosis heard mainly in cases of atrial septal defect is usually mid-diastolic in timing, blubbering in quality and apical in location. It is not influenced by respiration as a rule.

Relative tricuspid stenosis from dilatation of the right ventricle without necessarily increased tricuspid flow as in ASD has been reported by several writers (see Fig. 430 and p. 432 for such a case—a patient with primary pulmonary hypertension). In the interesting case of Rivero Carvallo *et al* (1279) there were both a rumbling mid-diastolic murmur and a presystolic crescendo. Furthermore the murmur was accentuated by inspiration as in organic tricuspid stenosis. MacCallum (1003) described a case from the Johns Hopkins Hospital, a 29 year old woman (J. H. Unit 11742) with obliterative lesions of the pulmonary arteries (probably multiple pulmonary

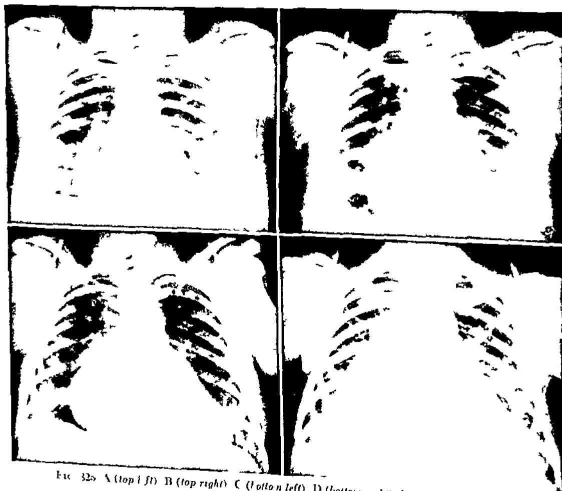


Fig. 325. A (*top left*), B (*top right*), C (*bottom left*), D (*bottom right*). See legend Fig. 325 E.

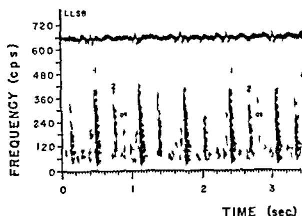


Fig 323 Tricuspid stenosis

Over lower sternum in J D (339660) 22 years old who has had repeated attacks of rheumatic fever There are signs of both aortic and mitral involvement but those of tricuspid disease are especially striking giant *a* waves in the jugular pulse and presystolic murmur with thrill over the lower sternum accentuated by or present only during inspiration *Large right atrium The presystolic* murmur was accentuated by inspiration and slightly *separated from* S_1 *The frequency* span of the presystolic murmur was greater than is usually seen in cases of MS The opening snap is probably tricuspid it shows a greater S OS interval than in the case of mitral OS and in records taken with breathing is virtually absent in expiration

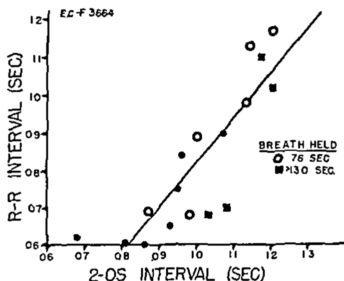


Fig 324 Tricuspid stenosis—interval between S_2 and tricuspid opening snap All measurements from one recording Atrial fibrillation Holding the breath had little effect (From Kossmann (816))

sound In fact the first sound has been essentially absent over the lower sternum in some cases (*e g* Fig 322) The tricuspid opening snap is rarely as close to the second sound as is its mitral counterpart Mainly this is because pressure in the right atrium rarely attains as high a level as *in the left atrium, probably because the* systemic venous compartment is so much larger than the pulmonary venous compartment The right *atrium is more distensible than the left* has different pressure volume characteristic In small part, the greater delay may be apparent rather than real, *one must relate the tricuspid* opening snap to the later pulmonary component of the second sound, not to the aortic component The S_2 to tricuspid OS *interval is likely to vary* with respiration, being shortest with inspiration for reasons identical to those for the exaggerated diastolic murmur with inspiration

The diastolic murmur of tricuspid stenosis has the same two components—early passive and late atriosystolic—as that of mitral stenosis It differs from the murmur of mitral stenosis in several respects *(1) It is likely to show impressive* variability with phase of respiration, being loudest in late inspiration or in the period of so called *inspiratory apnea (held inspiration) This phenom*enon is sometimes called Carvallo s sign (4 1276, 1277) As a rule the murmur of mitral stenosis shows *little respiratory variation if it varies at* all it is likely to be loudest in expiration However, I have encountered at least two instances of diastolic murmurs clearly of mitral origin, which were accentuated on inspiration Similarly, Schilder and *Harvey (1357)* noted that although the murmur of mitral regurgitation is likely to decrease with inspiration both the opening snap *and the diastolic rumble of mitral stenosis may* be increased They state that a number of patients have been observed who demonstrated *this Furthermore the murmur of tricuspid* stenosis of most severe degree is uninfluenced by respiration Apparently venous pressure is already so elevated that the increment produced by inspiration is inconsequential in its effects

(2) The murmur of tricuspid stenosis tends to be louder, to be accompanied by a striking thrill and to have a higher frequency span in the spectral *phonocardiogram than the murmur of mitral* stenosis The reason for these three intimately related phenomena is probably the more superficial position of the generator area in the case of the tricuspid valve The greater frequency range

of the murmur in tricuspid stenosis evident in Figure 322 and 323 cannot be taken as an indication that the murmur *would impress the ear* as being higher pitched. Probably the greater intensity of the components at lower frequencies outweighs the high frequency components. However Chesterman and Whitaker (258) state that *the diastolic murmur may* be high pitched and at times have a sea gull quality. This I have not observed.

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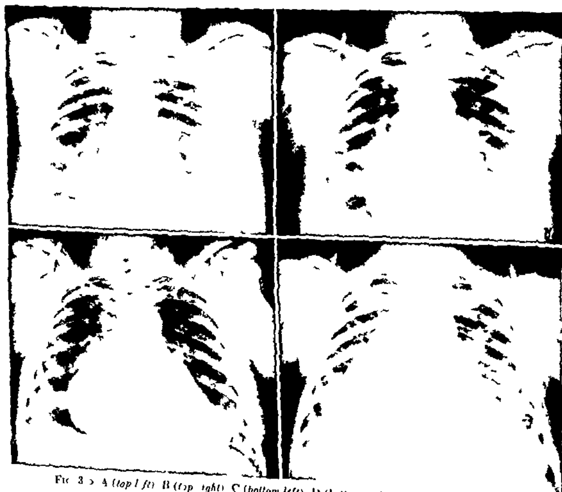


Fig. 3[illegible]. A (*top left*), B (*top right*), C (*bottom left*), D (*bottom right*). See legend Fig. 3[illegible].

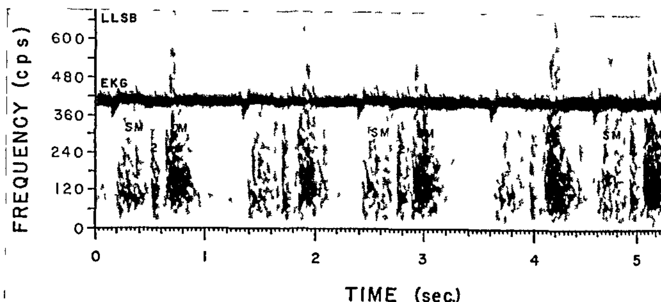


FIG. 325 E. Tricuspid stenosis.

C. D. (127526) 41 year old female has been seen at this hospital since 1933 when a chest x-ray already showed fullness of the right atrium consistent with tricuspid disease (A). This showed steady increase in the films taken in 1948 (*B*), 1952 (*C*) and 1957 (*D*). There is atrial fibrillation, enormous distension of the neck veins and liver, a striking diastolic rumble accompanied by thrill in the left midprecordium. *E* presents sounds at LLSB. Distinguishing this murmur from that of mitral stenosis is the large frequency span. As in the case displayed in Figure 3[illegible] the first heart sound is faint.

emboli) and a mid diastolic rumble at the apex. In 1935 Wyckoff and Bunim (1595) described three such cases and found ten in the literature. One of their cases was a 21 year old Puerto Rican woman seen in New York with cor pulmonale due to Schistosoma mansoni. They emphasized that the diastolic rumble could be quite constant, it was heard consistently over a period of seven years.

White (1539) wrote in 1947: "Functional tricuspid stenosis has not been described although I have recently encountered two cases which I believe to be such with well localized mid diastolic murmurs near the lower end of the sternum, mitral diastolic murmurs and loud pulmonary diastolic murmurs." He was apparently discussing cases of rheumatic heart disease. It is entirely possible that some of the instances of what has been interpreted as organic tricuspid stenosis with mitral stenosis have largely or only relative tricuspid stenosis. Reubi, Vogt and Plancherel (1263) described the case of a patient with panarteritis and pulmonary hypertension who had signs of pulmonary regurgitation and right sided hypertrophy. In addition there was at the apex a diastolic murmur suggesting mitral stenosis but interpreted by the authors as the murmur of relative tricuspid stenosis. Davis and Andrus (336) described a case of mediastinal collagenosis in which the pulmonary veins were severely constricted and a rumbling apical diastolic murmur produced probably through the mechanism of relative tricuspid stenosis. Brachfeld *et al.* (147A) described two patients with primary pulmonary hypertension and a diastolic rumble of relative *tricuspid* stenosis.

A right sided Austin Flint murmur may occur in cases of pulmonary hypertension with pulmonary regurgitation. I have observed three such instances. These are cases in which a conspicuous presystolic concentration of the diastolic murmur (of pulmonary regurgitation) at the lower left sternal border is demonstrated.

A sound suspiciously suggestive of an opening snap is even seen in some cases of the same type as may have the murmur of relative tricuspid stenosis, although not necessarily in association with a murmur.

TRICUSPID REGURGITATION (TR)

(Syn. Tricuspid insufficiency or incompetence)

ETIOLOGIC, ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS (935, 1133). An organic cuspal basis

is rather uncommon being of rheumatic or congenital origin when it occurs relative tricuspid insufficiency on bases similar to those outlined for the mitral valve (p 314) e g right ventricular failure (813 964) is common Morphine addicts ("mainliners") are prone to bacterial endocarditis affecting the tricuspid valve

The anatomic and physiologic changes are similar to those for the mitral valve Relative insufficiency occurs more readily than in the case of the mitral valve

CARDIOVASCULAR SOUND The systolic murmur of tricuspid regurgitation differs from that of mitral regurgitation mainly in its location of maximum audibility and in its change with respiration Both however are not invariable features distinguishing this lesion from mitral stenosis

The holosystolic murmur of tricuspid regurgitation is usually loudest in the left midprecordium Rarely is it *loudest* in the tricuspid area Dilatation of the right ventricle which occurs either as the cause or the result of the tricuspid regurgitation brings the right ventricle unusually far to the left Often in cases of mitral stenosis a loud systolic murmur at the apex is attributed to the association of significant mitral *regurgitation* However if surgery is performed it may be found that there is no regurgitant jet Furthermore with surgical relief of the mitral obstruction the systolic murmur may disappear This sequence of events suggests that right sided heart failure with relative tricuspid insufficiency produced the systolic murmur well heard at the apex and falsely interpreted as being caused by mitral regurgitation (1337 1482) With the onset of atrial fibrillation the evidences of tricuspid regurgitation in association with right ventricular failure may be especially striking In one 33 year-old patient (L A 70 884) who had giant *a* waves in the venous pulse and a presystolic pulsation of the liver as well as auscultatory signs of tricuspid stenosis but no evidence of tricuspid *regurgitation* striking *systolic* pulsation of the liver appeared in the last week of life with the development of atrial fibrillation and pronounced heart failure

The systolic murmur of TR may be accentuated by *inspiration* (1278) through an increase in venous return and therefore *in right ventricular* stroke volume However as compared with tricuspid stenosis the influence of respiration on the murmur of tricuspid *regurgitation* is less impressive Furthermore with right ventricular failure which is usually present the ventricle is already *operating* at maximum capacity and cannot increase its cardiac output significantly

In severe tricuspid regurgitation a loud murmur related to ventricular systole may be strikingly

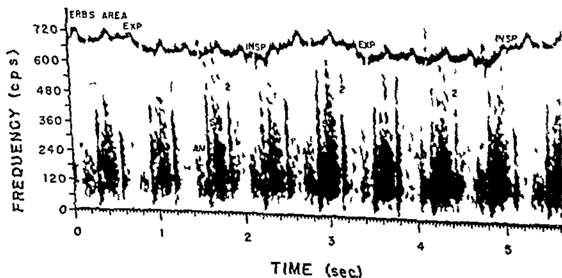


FIG 378 Tricuspid stenosis

I M (75655) [illegible] year old female has typical murmurs of AS and AR Tricuspid stenosis is indicated by the prominence of the presystolic murmur at Erb s area (shown here) its early occurrence with slight separation from S_1 despite a normal PR interval and its exaggeration with inspiration

audible over the veins of the extremities I am told (840) of an instance in which the murmur was so impressive in the region of the femoral triangle that, in combination with the tumescence of engorged veins in that area, arteriovenous fistula was misdiagnosed

PULMONARY STENOSIS (PS)

Rheumatism affects the pulmonary valve very rarely The overwhelming majority of cases of PS have a congenital basis However, as part of carcinoid cardiovascular disease, there may be acquired PS, combined usually with pulmonary regurgitation and with both types of functional defect at the tricuspid valve as well

Relative PS is frequent The most striking example is atrial septal defect in which there may not only be a striking systolic murmur but also a pressure drop of as much as 40 mm across the pulmonary valve Voluminous right ventricular outflow and dilation of the outflow tract above and below the valve are responsible Just as in the case of aortic stenosis the differentiation of marked relative from mild organic pulmonary stenosis may be difficult

So rare is any variety of PS except congenital that reference is made to the discussion of congenital pulmonary stenosis on pp 365–375

PULMONARY REGURGITATION[7]

ANATOMIC ETIOLOGIC AND PHYSIOLOGIC CONSIDERATIONS Relative pulmonary insufficiency secondary to pulmonary hypertension with production of the Graham Steell murmur is not uncommon (995) Actual rheumatic damage to the pulmonary cusps is rare The gonococcus has a notorious propensity to localization on the pulmonic valve *at least the gonococcus attacks* the pulmonic valve more frequently than most other organisms although when all cases of gonococcal endocarditis are analyzed involvement of the valves on the left side of the heart occurs more commonly than pulmonic involvement Pulmonary regurgitation along with other valvular defects can occur in carcinoid cardiovascular disease Trauma may result in pulmonary regurgitation Having enumerated these conditions all rare except for the Graham Steell murmur, the *acquired* types of pulmonary regurgitation have been nearly, if not completely, exhausted

Isolated pulmonary regurgitation on a congenital basis is likewise uncommon As was known to Leonardo, a quadricuspid semilunar valve is from the engineering standpoint, not as strong as a tricuspid valve Pulmonary regurgitation has been reported with quadricuspid valve (798) and with bicuspid valve (352A)

Pulmonary regurgitation relative or organic occurs with congenital malformations such as pulmonary stenosis, Eisenmenger complex ventricular septal defect, idiopathic dilatation of the pulmonary artery etc Isolated pulmonary regurgitation presumably congenital has been diagnosed in life by clinical and physiologic means by Kjellberg *et al* (800) Ford (469) and Morton and Stern (1117) Absence of the pulmonary valve with pulmonary regurgitation has been reported (229) the patient also had a ventricular septal defect The pulmonary valve may be absent in cases of tetralogy of Fallot with infundibular stenosis

With pronounced pulmonary regurgitation there is likely to be a striking hilar dance on fluoroscopy and by electrokymograms or roentgen kymograms large excursions of border movement in the region of the main pulmonary artery and rapid rise and fall of the limbs of the curves By cardiac catheterization there is usually a wide pulmonary arterial pulse pressure Particularly diagnostic is the steep slope of the dicrotic limb with fall of pressure in diastole to a level almost equal that in the right ventricle (22 1117)

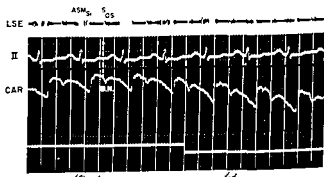


FIG 327 Proved tricuspid stenosis Note increase in presystolic murmur (ASM) with inspiration S_1 = first sound S_2 = second sound OS = opening snap (From Harvey (652))

[7] See reference 1067

Ellison and colleagues (425) found that the ejection volume was increased sufficiently in their dogs to produce a systolic gradient of as much as 40 mm Hg across the valve orifice in the absence of organic obstruction. The same gradient has been found in man (22, 469).

Pulmonary regurgitation is a well tolerated valve lesion. Barger and colleagues (52) and Ellison and colleagues (425) could not produce heart failure in dogs by destruction of the pulmonary valve. On the other hand Kay and Thomas (773) had one dog that died of right sided heart failure and thought that others would have done so if exercised. They attributed the difference in results to more complete excision of the pulmonary cusps. Oleson and Fabricius (1152) found that a patient had little incapacitation from pulmonary regurgitation of severe proportions 27 years after gonococcal endocarditis. Campeau and colleagues (229) describe a 52 year old with no pulmonary valve and death which was adequately accounted for by the associated lesion—VSD and single coronary artery with myocardial infarction.

CARDIOVASCULAR SOUND. *The Graham Steell murmur*, because of the high pressure in the pulmonary artery often matching or exceeding that in the aorta, is high pitched and resembles the murmur of aortic regurgitation precisely. It may be accentuated by inspiration (W. 599694)—a feature which does not occur often enough to be too helpful.

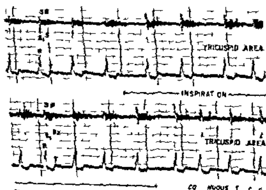


FIG. 328. Murmur of tricuspid regurgitation exaggerated by inspiration. This patient had surgically proved pure mitral stenosis. The systolic murmur could easily have been misinterpreted as indicating mitral regurgitation. (Courtesy of Harvey (659) and Circulation.)

In organic pulmonary regurgitation the diagnosis may be suggested by the fact that although the murmur begins immediately with the second heart sound it is relatively low pitched. When the murmur is generated at normal levels of pulmonary artery pressure (about 20 mm Hg at the time of pulmonary valve closure) *a murmur of quite different quality is produced* as compared with the situation when there is pulmonary hypertension.

Holldack and his colleagues (701, 1102) were impressed with the late *onset* or crescendo-decrescendo pattern of the diastolic *murmur of pulmonary regurgitation*. They describe with phonocardiogram cases of pulmonary regurgitation and an interval of 0.03 to .10 sec between the second sound and the onset of the murmur. In experimentally produced pulmonary regurgitation in dogs Rogers and his colleagues (1307) found a crescendo-decrescendo *pattern to the diastolic murmur or a gap between the second sound and the onset of the diastolic murmur.*

In a patient (C. S. M. 252820) who appears to have ventricular septal defect with *pulmonary hypertension the only* murmur is a long, rough diastolic murmur and thrill produced probably by pulmonary regurgitation. It is non-decrescendo. *Systole and diastole* have been of approximately equal length. Experienced observers have repeatedly mistaken it *for a systolic* murmur. *Holldack and colleagues (702)* described a very similar murmur in a patient with patent ductus arteriosus and pulmonary hypertension.

Davidson (330A) *described* a holodiastolic murmur with atrial septal defect and pulmonary hypertension. A diastolic thrill was palpable in one case.

LESS COMMON VARIETIES OF ACQUIRED VALVULAR HEART DISEASE

Bacterial endocarditis will be discussed later (pp. 450–452).

In gout tophi may *occur* on the mitral valve. The most acceptable case is that of Bunim and McEwen (190). *The* posterior mitral leaflet was the site of a large *urate* tophus. In life no murmur was heard. This is *probably* not surprising since the anterior (*or* aortic) leaflet alone is often adequate for closing the mitral orifice. Furthermore the tophus was located *in* the belly rather

than at the margin of this leaflet I have observed a 52 year old patient (P L, 692250) with extremely severe tophaceous gout and a loud mitral systolic murmur of obscure cause

In *ochronosis* (alkaptonuria), there may be deposit of the anomalous material not only in the intervertebral disks, leading to calcification and ankylosing spondylitis, but also in the heart valves, with production of calcific aortic stenosis (945A) In one case the authors stated "The aortic valve was stenotic and also showed extensive pigmentation and nodular calcification" (945A)

With *carcinoid tumors* (argentaffinomas) of the small bowel, especially with metastases in the liver or other sites, such as the ovary, which have direct venous drainage to the right side of the heart, there occurs damage to the tricuspid and



FIG 329 Appearance of patient described by Sir Maurice Cassidy (254) probably the first reported instance of *carcinoid cardiovascular disease* The flushing and the telangiectases in the butterfly area of the face are well shown The original in color well demonstrates patchy cyanosis over the upper part of the chest

pulmonary valves with stenosis and regurgitation (Figs 329 to 332) The precise mechanism is not clear However, something elaborated by the tumor and partially destroyed in the liver and lung seems to be responsible In one of the cases I reported (1068) the foramen ovale had been secondarily opened as a result of pulmonary hypertension the right sided valve lesion and right sided failure This patient developed not only polycythemia but also changes in the mitral valve and to some extent the aortic

A few instances of mitral stenosis in association with giant cell aortitis of the temporal arteritis type have been described (1252) The significance of the association is unclear

With impressive frequency aortic regurgitation is associated with Marie-Strumpell *spondylitis* Schilder and colleagues (1358) found spondylitis in five cases of aortic regurgitation Since spondylitis occurs with overwhelming preponderance in males one should consider particularly the incidence of spondylitis in male patients with aortic regurgitation 8 3 per cent The regurgitation was severe leading to death in three patients during the period of observation Histologically there was extensive change in all three layers of the base of the aorta Blumberg and Ragan (121) found six cases of valvular heart disease among 128 cases of rheumatoid spondylitis Changes in the heart valves especially in the mitral and usually located midway between the margin and base are found commonly in cases of peripheral rheumatoid arthritis which come to autopsy The changes resemble those of the rheumatoid nodule both grossly and microscopically Usually the lesions are of limited functional significance However Baggenstoss and Rosenberg (41) in a report which is one of the earliest on the subject



FIG 330 Liver metastases in a case with *carcinoid cardiovascular disease* × 100



FIG. 331. The gross appearance of the heart valves in a patient with *carcinoid cardiovascular disease*. (*Upper left*) Tricuspid valve. Note the sclerosis of the endocardium of the right atrium and perforation of the interatrial septum. (*Upper right*) Outflow tract of right ventricle with scarred pulmonary artery above, scarred tricuspid valve below. (*Lower left*) Close up of scarred pulmonary valve. (*Lower right*) Scarred chordae tendineae of mitral valve. Scarring of the mitral and aortic valves occurred in this case probably because of passage of blood to the left side via the atrial septal defect without circulation through the lung.

of the heart in rheumatoid arthritis described a case of tight mitral stenosis with classical deforming rheumatoid arthritis. Bauer and colleagues (62) have written about the aortitis and aortic valve disease of rheumatoid arthritis. Clark, Kulka and Bauer (271) found valvular involvement in 4.8 per cent of 1000 patients with rheumatoid arthritis—2.2 per cent had aortic regurgitation alone, 1.4 per cent had aortic regurgitation in combination with mitral stenosis. Aortic regurgitation alone or in combination with another valve lesion was present in the entire 48 patients. The patients with valvular involvement were almost exclusively men. Involvement of the spine was present in 90 per cent and of the hands in 50 per cent. The incidence of uveitis (60 per cent) was higher than in rheumatoid arthritis in general. Angina pectoris, presumably from involvement of the coronary ostia as part of the rheumatoid aortitis, was frequent. In this respect

and others—such as the long course with rapid deterioration once failure sets in—close simulation of syphilitic heart disease is produced

Figure 333 shows the main autopsy finding (autopsy 21422) in a child with tuberous sclerosis. A *rhabdomyoma* bulged into the outflow tract of the right ventricle producing a loud systolic murmur at the left sternal border and right sided heart failure. A similar situation resulting from a fibroma of the left ventricle has been described (1059). Although these are not strictly valvular the murmurs and the pathologic physiology suggest a valvular defect

In *diffuse systemic sclerosis* (visceral scleroderma) there may be a basilar diastolic murmur (e.g. J. S. 720832). There are two possible mechanisms (1) pulmonary regurgitation from fibrosis of the lung and pulmonary hypertension (2) aortic regurgitation from a lesion of the valve

Intrusion of the dilated aortic root into the mitral orifice (1573) in hypertension, syphilis, dissecting aneurysm (504) or the Marfan syndrome is a theoretically possible cause of mitral diastolic murmur

Valvular involvement in some degree occurs in about 40 per cent of cases of *primary systemic amyloidosis* (1321). Occasionally (766, 1032, 1271) it is so severe as to produce important functional defect. In many other cases murmurs both systolic and diastolic occur

Diverticulum of the mitral valve Renwick and Stewart (1261) describe a 63 year old patient who in the last year of life went blind from endocarditis, developed a loud blowing mitral systolic murmur and went into congestive heart *failure* At autopsy the anterior mitral leaflet was distorted by a pouch large enough to accommodate the tip of the little finger to a depth of over 1 cm. The pouch was directed backwards so that during ventricular systole it would impinge on the posterior cusp as a round ball leaving a triangular orifice on either side. The pathogenesis was obscure

In *systemic lupus erythematosus* (658a) I have heard typical auscultatory signs of pure mitral stenosis in one case (A. V. 443557) and of mitral stenosis and regurgitation and aortic regurgitation in another (B. T. 420876). In a third patient (C. N. 317341) there is profound aortic regurgitation which in large part had its basis in two bouts of bacterial endocarditis complicating Libman-Sacks lesions of the aortic valve. Figure 334 presents a case of mitral stenosis due possibly to SLE. In 1924 in the classic article on a hitherto undescribed form of valvular and mural endocarditis Libman and Sacks (945) referred to the presence of a presystolic rumble in three cases. In one which came to post mortem examination thickening of the mitral valve but no Aschoff bodies were found. Griffith and Vural (6?3) found typical rheumatic mitral stenosis in a patient whose other clinical and pathologic features were those of lupus. Shearn and Pirofsky (1381) found a systolic murmur in 24 of 31 cases, a gallop in 7 of 31 and an apical diastolic murmur in 4 of 31. The diastolic murmur was presystolic in one and mid-diastolic *in three*. Both patients who came to post mortem examination displayed Libman-Sacks changes of the mitral valve. *Gibson and Wood (518) described the case* of a 33 year old man with presumed SLE and presumed isolated tricuspid stenosis. All clinical findings were consistent with these diagnoses but LE cells *were not identified*. The valvular involvement in SLE is more impressive *pathologically* than clinically. Involvement of the pulmonary and tricuspid valves is often more striking than that of their counterparts on the left side. Clinically an apical systolic murmur occurs in possibly half of the cases. Gallops occur *frequently*. Hypertension and its auscultatory changes *are* usually absent except with end stage renal involvement and/or with steroid therapy

Atherosclerosis so important in the pathology of the aortic valve frequently involves the ventricular aspect of the aortic leaflet of the mitral valves in a plaque distribution (31, 661, 1311). Rarely if ever is this involvement *per se* of hemodynamic, clinical or other significance (458)

With *calcification of the annulus fibrosus mitralis* a harsh systolic *murmur* may develop (1121). The murmur probably is on the basis of mitral regurgitation. This is a variety of relative insufficiency—interference with contraction of muscular component of the mitral ring is probably responsible. It must be *pointed out* that Fertman and Wolff (458) were unable to relate the occurrence of a murmur to calcification of the mitral annulus



FIG 331 The gross appearance of the heart valves in a patient with *carcinoid cardiovascular disease* (*Upper left*) Tricuspid valve Note the sclerosis of the endocardium of the right atrium and perforation of the interatrial septum (*Upper right*) Outflow tract of right ventricle with scarred pulmonary artery above scarred tricuspid valve below (*Lower left*) Close up of scarred pulmonary valve (*Lower right*) Scarred chordae tendineae of mitral valve Scarring of the mitral and aortic valve occurred in this case probably because of passage of blood to the left side via the atrial septal defect without circulation through the lung

of the heart in rheumatoid arthritis described a case of tight mitral stenosis with classical deforming rheumatoid arthritis Bauer and colleagues (62) have written about the aortitis and aortic valve disease of rheumatoid arthritis Clark, Kulka and Bauer (271) found valvular involvement in 4.8 per cent of 1000 patients with rheumatoid arthritis—2.2 per cent had aortic regurgitation alone 1.4 per cent had aortic regurgitation in combination with mitral stenosis aortic regurgitation alone or in combination with another valve lesion was present in the entire 48 patients The patients with valvular involvement were almost exclusively men Involvement of the spine was present in 90 per cent and of the hands in 50 per cent The incidence of uveitis (60 per cent) was higher than in rheumatoid arthritis in general Angina pectoris presumably from involvement of the coronary ostia as part of the rheumatoid aortitis was frequent In this respect



Fig 332 H

Fig 332 Carcinoid cardiovascular disease Microscopic appearance of the valves in the same case as illustrated in the last figure A Tricuspid valve × 4 B Pulmonary valve × 4 C Mitral valve × 4 D A mitral chorda tendinea × 8 E Aortic cusp × 23 F Normal aortic cusp from adult for comparison × 23 G Bulbous tip of tricuspid valve Ventricular surface above atrial aspect below Hyaline avascular and acellular material is present on the atrial side of the valve × 75 H Striking evidence of current activity of inflammatory process × 100



Fig 333 *Rhabdomyoma* in outflow tract of right ventricle loud systolic murmur The child had tuberous sclerosis



Fig 332 *A* (*left*) and *B* (*right*)

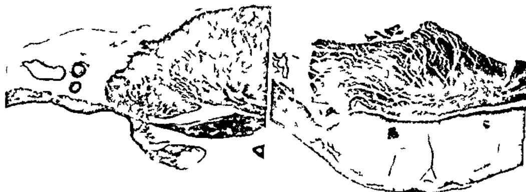


Fig 332 *C* (*left*) and *D* (*right*)



Fig 332 *E* (*left*) and *F* (*right*)



Fig 332 *G*



FIG 332 H

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FIG 333 *Rhabdomyoma* in outflow tract of right ventricle loud systolic murmur The child had tuberous sclerosis

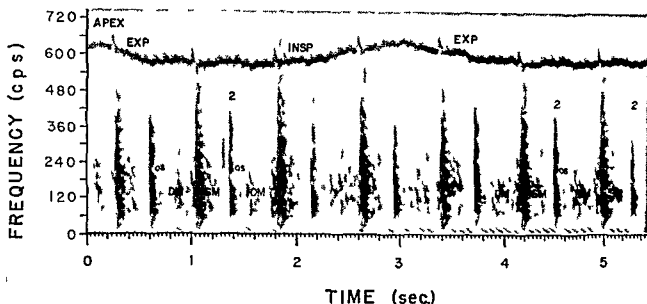


Fig 334 Probable mitral stenosis in patient with chronic polyserositis possibly SLE
I B T (325984) now age 43 had a butterfly rash and a chronic illness manifested by repeated rapid accumulations of peritoneal and pleural fluid requiring frequent paracentese In recent times fluid accumulation has been controlled by salt restriction and mercurial diuretics However auscultatory signs of mitral stenosis have appeared On cardiac catheterization performed at the time of this recording the pulmonary capillary pressure at rest was normal The recording shows undoubted signs of mitral stenosis snapping M_1 opening snap diastolic rumble with presystolic crescendo M_1 is delayed very little

Blackman (110) reported on *syphilis of the mitral valve* resulting in mitral regurgitation

With *endomyocardial fibrosis* (not to be confused with fetal fibroelastosis) the valve mechanism—in the case of the tricuspid or the mitral or both—often is so affected that a systolic murmur which is often high pitched and may be accompanied by a thrill, is produced (332) Adhesion of the posterior mitral cusp with immobilization is a frequent basis of mitral regurgitation This disease occurs with extraordinarily high frequency in the natives of some parts of Africa (47) Diastolic murmurs were unimpressive both in incidence and intensity in the 20 cases reported by Ball Davies and Williams (47) from Uganda

During *insulin shock therapy* a wide pulse pressure is likely to be observed and a basilar diastolic murmur may be heard Although I have not studied the matter systematically, I am told by a keen observer of a patient (J K, 710776) who with insulin administration developed "acute aortic insufficiency" with "zero diastolic pressure" and an aortic diastolic murmur With glucose administration both of these manifestations disappeared in a few minutes Mielke and Holldack (1102) have made similar observations They claim to reproduce the entire picture, including the basilar diastolic murmur, with epinephrine administration and suggest that this is the mechanism of the phenomenon with insulin shock They further suggest that the pulmonary valve is the source of the murmur mainly because the murmur displayed characteristics they ascribe to that of pulmonary regurgitation—crescendo decrescendo pattern or more often, a brief gap between S_2 and the onset of the murmur

CHAPTER 16

Congenital Cardiovascular Disease

The order in which the various congenital malformations will be discussed has been chosen not on the basis of any complicated embryologic or other system of classification but rather with a mind to eliminating repetition as much as possible

ATRIAL SEPTAL DEFECT (ASD)

(Syn Interatrial septal defect [IASD] interauricular septal defect patent foramen ovale etc)

DEFINITION Any permanent patency of a portion of the interatrial septum

ANATOMIC CONSIDERATIONS (719) Exclusive of mere flap patency of the foramen ovale which is present in about 16 per cent of cases (719) and will not concern us here two anatomical types are recognized (1) Atrial septal defects of the persistent ostium secundum type are located relatively high in the septum in the area of the fossa ovalis and there is usually no involvement of the atrioventricular valves The secundum type of atrial septal defect is anatomically hemodynamically and clinically the classical one and the one most amenable to presently available methods for surgical repair (2) With persistence of the ostium primum the defect is located low in the atrial septum i e close to the atrioventricular ring and abnormality of the tricuspid or mitral valve with regurgitation at one of these valves is likely to occur This second type constituting 10 to 15 per cent of all cases of ASD (228) is a variety of endocardial cushion defect (see p 350)

Frequently shreds of septal remnants persist across the septal defect (Fig 335) (580 Figs V 2 and II 44 719)

[illegible] septal defect

The other anatomical findings are consequences of the shunt The right atrium ventricle and pulmonary artery are usually greatly dilated Hypertrophy also occurs The enlargement of the right side of the heart tends to produce rotation of the heart in a clockwise direction (viewing the heart from the apex) The right ventricle comes to occupy the cardiac apex and to extend further to the left than is ordinarily the case The left atrium left ventricle and aorta are usually small

Lutembacher's syndrome (997 1061)—ASD with mitral stenosis (Fig 335)—is rarer than is often thought Among 2,000 autopsies (1138) there were 87 cases of ASD of which 5 had associated *mitral stenosis*

PHYSIOLOGIC CONSIDERATIONS In uncomplicated ASD the net shunt is from the left atrium to the right atrium It is necessary to speak of *net* shunt because studies—particularly with injection of dye into a vein of a lower extremity demonstrate that there is often a small right-to-left shunt involving inferior vena cava blood This right-to-left shunt usually is not sufficient to produce a degree of oxygen unsaturation of arterial blood below what is accepted as the lower limit of the normal range

The magnitude of the shunt may be such that the right ventricle pumps considerably more blood than the left ventricle Pemberton (1199) found as have others that systemic flow is normal in most cases of ASD In his cases pulmonary flow exceeded systemic flow by 36 to 78 per cent (average 60 per cent) others have found pulmonary flows more than twice normal When the volume of the shunt is very large the arteriovenous oxygen difference becomes so small as to fall within the range of error for the method A small deviation in the determination of pulmonary



FIG 335 Remnant of atrial septum in case of large secundum defect Viewed from left side in V B (406046) 33 year old white female with Lutembacher's syndrome The mitral stenosis was rheumatic The scarred mitral valve is visible in the lower portion of the photograph

artery oxygen saturation results in a large difference in the flow value calculated

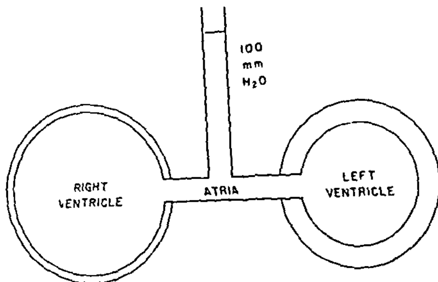
In the past, various theories have been advanced to account for the left to right shunt (1) Normally the pressure in the left atrium is slightly higher than that in the right atrium However, with an ASD of clinically significant size the two atria are fundamentally one chamber and by the laws of physics pressure must be identical in all parts of this chamber (2) Since the left atrium usually is superiorly located in relation to the right atrium, it has been suggested (1480) that by gravity blood tends to fall into the right atrium from the left atrium This suggestion was put to critical test by Brannon, Weems and Warren *(160), who could demonstrate* no change in the direction or degree of the shunt with changes in position

(3) The most credible possibility is that the left to right shunt is related to the difference in pressure volume characteristics of the two ventricles (351, 369, 723, 1029) The concept can be represented by the diagram in Figure 336 The two ventricles are represented as balloons, one (the "right ventricle") thin walled and one (the "left ventricle") thick walled, connected to a common reservoir and pressure head, represented as a T tube Because of the less steep pressure volume curve of the right ventricle,[2] an expression of its greater distensibility, the right ventricle accepts the larger volume of blood from the common reservoir, as would be predicted from *the simplified model with balloons (Fig 336)* In accordance with this concept, it is significant that the left to right shunt occurs mainly during ventricular diastole Therefore, the systolic murmur is not likely to be generated at the septal defect itself[3] This theory does not exclude the possibility that some blood may be transferred from the left atrium to the right atrium even during ventricular systole In fact this seems very likely for at least two reasons (1) the right atrium is more distensible than the left (Fig 337), (957), and (2) the right atrium becomes large in ASD The greater distensibility of the right atrium is predictable from the difference in the endocardial lining That of the left atrium is fairly fibrous giving a relatively smooth and pearly gross appearance that of the right atrium is thin so that the muscular trabeculations are clearly seen

To electrocardiographers and particularly in congenital heart disease the concept of systolic vs diastolic ventricular overload (210 211) is useful (1602) since each variety is usually attended by *a distinctive electrocardiographic pattern* In conditions of systolic overload of the right ventricle exemplified by pulmonary stenosis, there are simple evidences of ventricular hypertrophy right axis deviation in the standard limb leads and R waves of large amplitude in leads at right end of the precordial series In conditions

[2] *See reference 739A for comparative pressure volume* curve of the two ventricle

[3] Intracardiac phonocardiography in case of ASD reveals a systolic murmur in the outflow tract of the right ventricle but not in the atrium (1421)



PROBABLE MECHANISM OF SHUNT IN I A DEFECT

FIG 336 The difference in the pressure volume characteristics of the two ventricles is represented by balloons with different thicknesses of wall Both are connected to the same pressure head by a T tube Obviously the thin walled balloon dilates more

of diastolic overload of the right ventricle exemplified by the condition under discussion atrial septal defect the characteristic electrocardiographic finding is incomplete right bundle branch block Although usually the QRS does not attain or exceed 0 12 sec there is characteristically an RSR pattern in lead V_1 This finding does not occur in all cases of atrial septal defect as previously thought (ol 1137 p 293) but does occur in some two thirds of the cases Milnor and Bertrand (1107) found complete or incomplete bundle branch blocks in 17 of 24 proven cases of ASD The incidence may be higher if statistics are based on V_{3R} Complete right bundle branch block occurs uncommonly

In the ostium primum type of ASD there is often an associated mitral valve cleft with mitral regurgitation and strain on the left ventricle In the electrocardiogram the combination of left axis deviation with incomplete right bundle branch block is rather characteristic of ASD of the ostium primum type with associated cleft of the mitral valve

Pulmonary hypertension may develop in older patients with ASD because of wear and tear on the pulmonary vasculature (changes secondary to increased flow of long standing) At times pulmonary hypertension may be present early owing possibly to persistence of the fetal pattern of pulmonary vessels Wood (1591) has what appears to be a different view of the pulmonary hypertension of ASD and many other conditions He thinks that these individuals representing perhaps 20 per cent of all cases are persons whose pulmonary vasculature reacts in a vigorous and vicious manner to certain agents which do not ordinarily produce this effect unless present in extreme degree It is suggested that the behavior is congenital Wood found six cases of pulmonary hypertension (pulmonary pressure 43 to 81 mm Hg) among 50 cases of ASD The duration of the shunt he thought *could not be* held responsible since the youngest subject was 2½ years old and the average of the six was less than that of the rest of the group Welch and Kinney (1 24) in cases of ASD as well as patent ductus *arteriosus and* ventricular septal defect were *unable* to get evidence in support of the wear and tear effects of high flow

The dilatation of the pulmonary artery is secondary to the increased pulmonary flow and is not necessarily dependent on the existence of pulmonary hypertension There is usually a pressure gradient across the pulmonary valve This

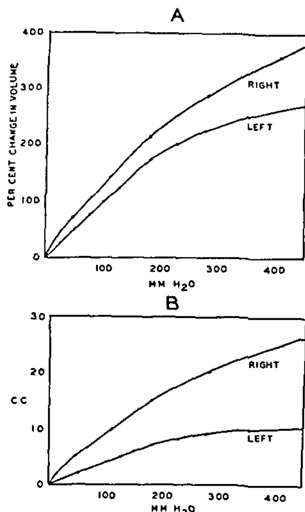


Fig. 337. Pressure volume characteristics of atria. *A*, Relative curves. *B*, Absolute curves. (From Little (957).)

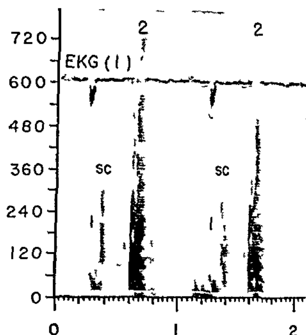


Fig. 338. Pulmonary area with *atrial septal defect*. *The first heart sound is virtually absent. The conspicuous early systolic click was confused for an unusually snappy* S_1. S_2 is split; its second component (pulmonary closure) is greatly accentuated. The early systolic click *initiates a soft systolic murmur. A faint early diastolic* murmur follows the accentuated pulmonary closure sound.

is interpreted as representing relative pulmonary stenosis. There may be as much of a discrepancy as 35 mm. Hg or even more (1061A) between systolic pressure in the right ventricle and pulmonary artery. The pressure loss in these cases is probably represented by gain in velocity, i.e., there is a conversion of pressure energy to kinetic energy. Normally, kinetic energy may represent as little as 5 per cent of the total made available by the contracting ventricle. In cases of ASD the figure may be several times this value. Indirectly the high value for kinetic energy is related to the systolic murmur which is produced in the outflow tract and/or pulmonary artery. Therefore the pressure gradient across the pulmonary valve should bear some relationship to the intensity of the murmur.

CARDIOVASCULAR SOUND. Atrial septal defect is to the group of congenital malformations what mitral stenosis is to acquired valvular lesions. In all their ramifications the auscultatory phenomena illustrate principles which are pertinent in connection with most forms of congenital heart disease. The features requiring discussion are at least ten in number:

1. The tricuspid diastolic rumble
2. The pulmonary systolic murmur
3. The pulmonary early systolic click
4. The mid systolic click
5. The pulmonary early diastolic murmur
6. The split second heart sound
7. The split first heart sound
8. The tricuspid opening snap
9. The additional features of the ostium primum variety of ASD
10. The sonic phenomena which simulate those of mitral stenosis

A mid diastolic murmur may be present at the apex, which is usually the right ventricle in these cases, or some point between the apex and the left sternal border. It has the auscultatory charac

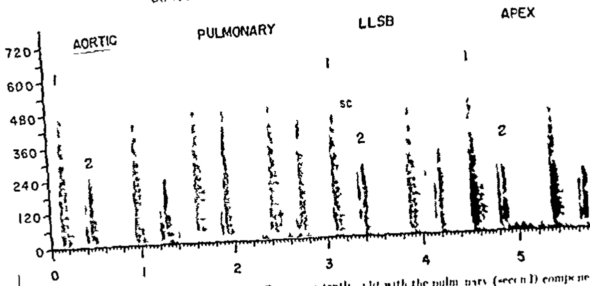


FIG 339 Atrial septal defect in V N (60624) S₂ is consistently split with the pulmonary (second) component very loud so that it is audible in both the aortic and the apical area Mid systolic click at LLSB probably extra cardiac S₁ is unusually loud

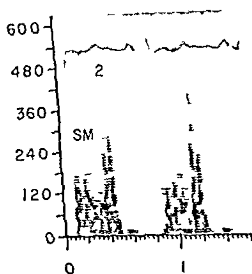


FIG 340 Split S₂ in ASD LLSB

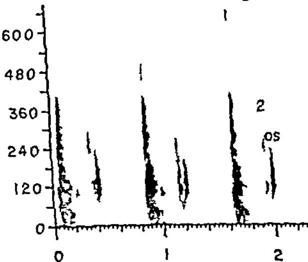


FIG 341 Mitral opening snap

For comparison with the split second sound shown in preceding figures S₂ followed by opening snap at apex in patient with mitral stenosis Note ringing M₁ Usually each component of a split S₂ has the appearance of a valvular closure sound with knobbed ridges for example Opening snaps have a purer frequency content and usually the frequency bottom does not extend to zero

teristics of a Carey Coombs murmur (p 199) Barber Magidson and Wood (51) found it to be audible in 9 of 62 cases of ASD—a third sound was present in 26 Blount and colleagues (120) described the disappearance of a mid diastolic murmur at the fourth left intercostal space after complete surgical closure of the defect There is usually no presystolic accentuation The murmur is explicable on the basis of the combination of high flow across the tricuspid valve (what Wood (1689) has termed torrential flow) and dilatation of the right ventricle Pree and colleagues (1234) call it a hypervolemic murmur Nadas (1137 p 280) states the rule of thumb that to get a diastolic rumble the pulmonary flow must be at least twice systemic flow Presumably he

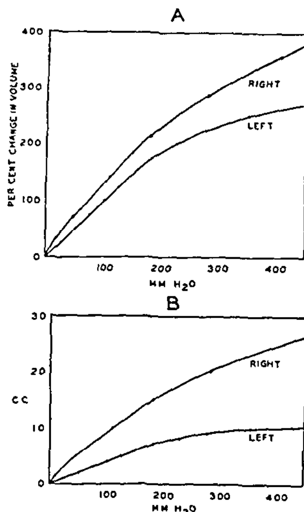


Fig 337 Pressure volume characteristics of atria A Relative curves *B* Absolute curves (From Little (957))

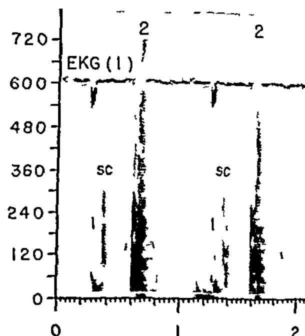


Fig 338 Pulmonary area with *atrial septal defect* The first heart sound is virtually absent The conspicuous early systolic click was confused for an unusually snappy S_1 S_2 is split its second component (pulmonary closure) is greatly accentuated The early systolic click initiates a soft systolic murmur A faint early diastolic murmur follows the accentuated pulmonary closure sound

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3 The pulmonary early systolic click
4 The mid systolic click
5 The pulmonary early diastolic murmur
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9 The additional features of the ostium primum variety of ASD
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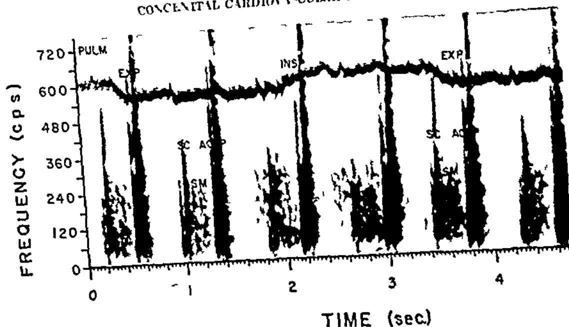


Fig 343 Atrial septal defect

Pulmonary area in D D (364143) 26 year-old female with long mi diagnosed rheumatic heart disease ECG within normal limits Features typical of ASD (1) Early systolic click maximal in expiration (2) decrescendo systolic murmur (3) widely split S

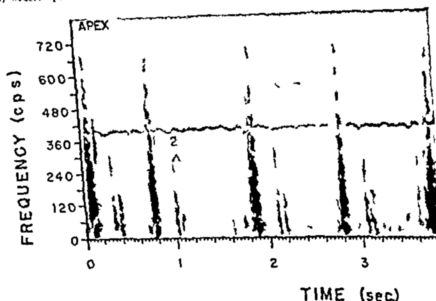


Fig 344 Variable S_2 splitting in ASD

P B (103156) 5 year old man was *proved by catheterization to have* ASD Atrial fibrillation was present *After* longer diastolic periods the degree of splitting of S_2 is greater

listen to the same patient and hear only so unimpressive a systolic murmur that the competence of the previous observer is questioned That the murmur indeed has origin in the pulmonary artery and not at the septal defect is supported by the physiologic considerations discussed above Furthermore Rushmer (1703 p 305) found that in dog with surgically created ASD the microphone found the *murmur* over the pulmonary artery not over the atria and by intracardiac phono

thinks this approximation holds for both the right side in ASD and anomalous venous return and the left side in VSD and PDA

It is possible that a mid diastolic murmur is sometimes generated at the septal defect The remnants of septal tissue, which sometimes traverses the defect, and the lip of remaining septum may be set into vibration Luisada (973) recorded *mid diastolic murmurs* over the base of the heart and attributed them to the mechanism mentioned

The pulmonary systolic murmur is the result of high flow through the outflow tract during ventricular ejection Its intensity is highly variable from patient to patient and at various times in the same patient For example, a patient may have a systolic thrill and systolic murmur at the left sternal border so loud that ventricular septal defect is considered At another time one may

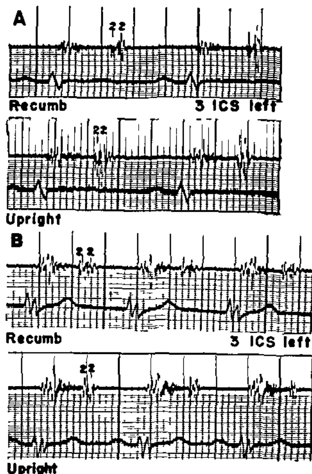


Fig 342A

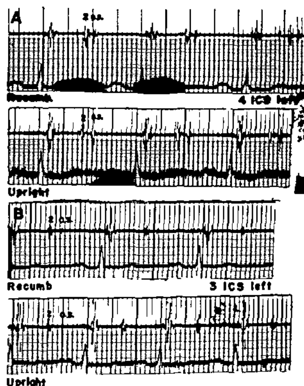


Fig 342B

Fig 342A *A* Decrease in splitting of P_2 of ASD when subject upright Phonocardiogram and lead II of the electrocardiogram of a 34 year old man with atrial septal defect The calculated left to right shunt was 6 62 L per minute and the right ventricular pressure was 40/8 mm Hg Note the wide splitting of the second sound (0 06 second) in the recumbent position and a decrease in splitting (0 03 second) in the upright position The heart rate is almost identical in both tracings *B* Phonocardiogram and lead II of the electrocardiogram of a 24 year old woman with atrial septal defect The calculated left to right shunt was 8 96 L per minute and the right ventricular pressure 42/10 mm Hg The electrocardiogram shows an incomplete right bundle branch block with QRS duration of 0 11 sec Note the wide splitting of the second sound (0 07 sec) in the recumbent position and a decrease in splitting (0 04 sec) in the upright position The heart rate is nearly identical in both tracing

Fig 342B *A* Increase in S_2 OS interval in MS when subject upright Phonocardiogram and lead II of the electrocardiogram of a 42 year old woman with rheumatic heart disease and mitral stenosis The calculated mitral valve area (Gorlin s formula) was 0 6 cm ^{2} and the pulmonary artery pressure 56/28 mm Hg Note that the duration of the interval between the second sound and the opening snap (OS) measures 0 06 sec in the recumbent and 0 09 sec in the upright position The heart rate is nearly the same on both occasions *B* Phonocardiogram and lead II of the electrocardiogram of a 43 year old woman with rheumatic heart disease and mitral stenosis Calculated mitral valve area = 1 2 cm ^{2} pulmonary artery pressure = 37/23 mm Hg Note that the duration of the interval between the second sound and the opening snap (OS) measures 0 09 sec in the recumbent and 0 11 sec in the upright position The heart rate is more rapid in the upright position Courtesy of Surawicz and *Circulation* (1451)

enormously dilated pulmonary artery coming into contact with the anterior chest wall, its vigorous pulsation producing the friction sound.

The split second heart sound is a highly characteristic feature of ASD. It was audible as grade III splitting in 52 of 62 cases (51). It has [illegible]

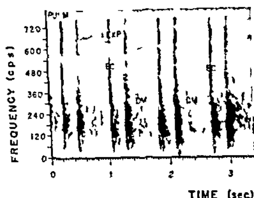


Fig. 346. ASD with pulmonary regurgitation.

Pulmonary area in F. W. (71881), 38 year old female with ASD and pulmonary hypertension. Record made six months after pulmonary denervation and three months after closure of the septal defect. The loud early systolic sound is too late for S_1 and must be early systolic click. S_2 is split with its second component tremendously accentuated and followed by a decrescendo murmur of pulmonary regurgitation. Cardiac catheterization showed no change in pulmonary pressure (about 80/20 mm. Hg) after the operations.

in the discrepant stroke volumes—and therefore systolic duration—of the two ventricles. Pulmonary valve closure is delayed in relation to aortic valve closure. It has long been appreciated that increase in venous return and increase in stroke volume prolongs mechanical systole of the ventricle (pp. 117 to 122). The split S_2 of ASD is a special, striking, persistent and pathologic variant of a phenomenon seen in most normal individuals—namely, splitting of the second sound with inspiration owing to the increase in venous return.

Since it is now clear earlier occurrence of aortic closure is responsible for part of the normal inspiratory splitting of S_2 (p. 150) the absence of change in the timing of aortic closure in cases of large ASD with fixed splitting of S_2 requires explanation. It is entirely plausible to presume that because of the congestion of the pulmonary circuit and the fact that the two atria are functionally one, inspiration will not result in the usual reduction in left ventricular filling in ASD. In some cases although the interval between the aortic and pulmonary components is fixed, both components fall slightly later in inspiration than in expiration (147). The explanation afforded is that the venous return from the lung is not reduced in these cases or if anything is increased (because of the pulmonary hyperemia) that the

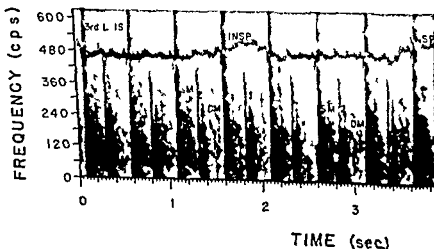


Fig. 347. ASD with pulmonary regurgitation.

Third left interspace in F. N. (B35[illegible]), a 6 year old female. S_1 is loud; there is a decrescendo ejection systolic murmur. S_2 is characteristically split and the second (pulmonary) component is followed by a decrescendo diastolic murmur. EKG was that of right ventricular hypertrophy, not right bundle branch block, in this case of ASD with pulmonary hypertension.

cardiography in patients with ASD Soulié *et al* (1421) found the murmur in the pulmonary artery, not in the atria

Transmission of the systolic murmur to the interscapular area of the back is a diagnostically helpful feature Usually the systolic murmur of ASD is about as loud in the back as anteriorly Coarctation is one of the few other conditions which show this phenomenon The murmur of pulmonary stenosis when very loud is, as one would predict, well heard in the back However, confusion is not likely to be created thereby

The early systolic click (Fig 338) represents a snapping of the pulmonary arterial wall early in ejection In no other single condition does it occur more consistently than in ASD By phonocardiograms it can be demonstrated in virtually all cases Its characteristics have been described in detail elsewhere (see p 179)

In one study a mid systolic click was recorded from the left sternal border in many cases It was attributed (1082) to movement of the costochondral and/or chondrosternal joints by the hypertrophied right ventricle, i e it was thought to be related to the left parasternal heave Since the finding has not been noted on stethoscopy and other phonocardiographic studies have not commented on it, there is a possibility that the mid systolic click is an artifact characteristic of the microphone or of the recording and analyzing set up used in this study, when exposed to the intense impulse of the hypertrophic right ventricle

A diminuendo pulmonary diastolic murmur (Figs 346 and 347) is heard occasionally In one series it was audible in 36 of 62 cases (51) but in most series the incidence has been less Bedford and colleagues (80) found it in 20 per cent I found it in 2 of 11 cases (1082), in one the murmur was accentuated *in inspiration* It has its basis in dilatation of the pulmonary ring is part of the general enlargement of the pulmonary artery It may persist after closure of the atrial septal defect, particularly in adults and in children with pulmonary hypertension

Coulshed and Littler (306) in describing five *instances of ASD in aged patients (58 to 79 years)* reported that in one an alleged pericardial friction rub persisted for five years! It had a superficial quality and became louder with pressure on the stethoscope Although it may be that systolic and diastolic murmurs were incorrectly interpreted as a pericardial friction rub the authors suggested that it may have been produced by the

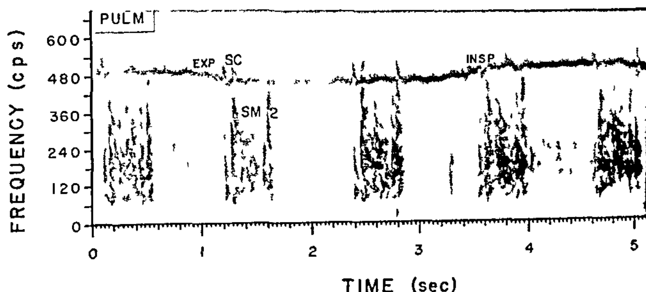


Fig 345 ASD with normal EKG

Pulmonary area in *J H B (750584) 27 years old with ASD (or AVR) proven by* cardiac catheterization but with normal EKG The findings are typical of ASD (or possibly AVR) (1) early systolic click followed by (2) a decrescendo systolic murmur and (3) a widely split S_2 *of which the second (pulmonary) component is* louder Splitting of S is exaggerated in inspiration The findings indicate that incomplete right bundle branch block is not invariable in ASD and that there may be respiratory variation in the splitting of S in cases of small ASD

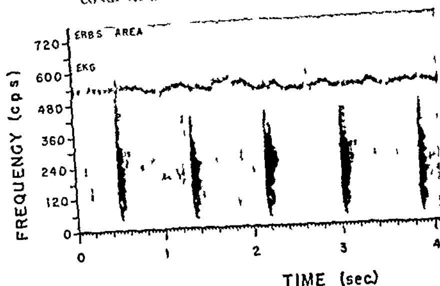


Fig. 349 ASD post-operative possible Lutembacher syndrome

R. F. S. K. (684[illegible] 60825) 30 year old had closure of an ASD $2\frac{1}{2}$ years previously. Catheterization failed to demonstrate any evidence of persistent shunt. The SPCG (Erb area) demonstrates persistent splitting of S which is however of minor degree and shows normal exaggeration with inspiration. There is an early diastolic sound with the characteristic of an AV opening snap. Furthermore it is probably of mitral origin since it maintains a constant relationship to the first (aortic) component of S_2. The mitral valve may be involved in this patient—Lutembacher's syndrome.

because the tricuspid valves are widely separated by the torrential tricuspid flow which is likely to continue through the entirety of diastole. The wider separation of the leaflets accounts not only for the delay in the sound of closure but also for the accentuated and ringing character which that sound frequently has. The combination of ringing tricuspid closure sound following a normal or somewhat reduced mitral closure *sound* gives a crescendic construction to the first sound (Fig. 348). This combination may simulate *the presystolic* murmur and ringing first sound of mitral stenosis.

Leatham and Gray (863) found a snap with the proper temporal relationships for an AV *opening* snap presumably a tricuspid opening snap. There are other instances (e.g. patent ductus and ventricular septal *defect) to indicate* that not only can a diastolic murmur of relative stenosis be attributed to torrential flow but also an opening snap. It is quite credible that even a *normal* valve might produce a snappy sound if opened vigorously.

One of the clinical clues to the presence of the ostium primum variety of ASD is the presence of the systolic murmur of mitral regurgitation with radiation to the left axilla. However the AV values may be normal we have *observed two such cases* (Z.O. 673149 B.S. 696242) in which the auscultatory findings *were no different from those* of the secundum variety of ASD (see Fig. 1[illegible]).

Lutembacher's syndrome—mitral stenosis (acquired or congenital) and ASD—is uncommon. An exaggerated impression of its incidence is created by a galaxy of auscultatory and other clinical features which suggest mitral stenosis in cases of pure ASD. Quite aside from the fact that the radiologic configuration of the heart (except for the absence of left atrial enlargement) and the EKG findings are often consistent with mitral stenosis there are the following some features mimicking mitral stenosis:

1. Crescendic first sound with accentuated *tricuspid* element suggesting presystolic murmur and ringing mitral first sound (see above).

2. *Split* second sound suggesting second sound proper plus mitral opening snap.

3. Mid-diastolic rumble—although of tricuspid origin—is often in the region of the apex (which in this condition *is likely* to be the right ventricle)

two ventricles are filled from a common reservoir, and that the effective filling pressure of both ventricles is increased in the manner that only the right ventricle is influenced normally during inspiration

In most cases of ASD (except those with relatively small shunts) the degree of splitting is constant ("fixed" is becoming the cliché) and displays no variation with respiration (Fig 344) There may be some reduction in the degree of splitting when the subject is in the standing position as compared with the recumbent position (1454) Marked variation in the degree of splitting with inspiration and posture suggests that the diagnosis is not ASD, or that the shunt, if present, is small The splitting of S_2 in ASD is not dependent on the presence of bundle branch block (Fig 345) complete or incomplete (although it may be increased with complete right branch block), or on the presence of pulmonary hypertension (although the splitting tends to be reduced in such cases) In fact with pulmonary hypertension the splitting may be less because the volume of the left to right shunt is reduced (1591)

Seemingly no one has attempted a systematic correlation of the volume of the shunt (or of the discrepancy between pulmonary and systemic flows) with the degree of splitting Splitting of S_2 was not found by Bertrand et al (96) in dogs with experimentally produced ASD, Rogers et al (1307) found it some weeks after creation of the defect

Blount and colleagues (120) observed disappearance of fixed splitting after surgical closure of the defect Wood (1591A) and many others have made similar observations Wood states the splitting may still be present when the subject is recumbent but not when he is upright He thinks reduced resistance to right ventricular filling may persist for a time after operation

Intensity of S_2 is not a reliable index of the level of pulmonary pressure since, as in other disorders leading to dilatation of the pulmonary artery, intensification appears to be related to proximity of the vessel to the anterior chest wall P_2 is commonly loud in ASD, regardless of the level of pulmonary artery pressure Wide separation of the cusps just before closure, as a result of the high flow, may be another factor in the loud P_2 of ASD

The first heart sound is also split at times (Fig 154), although this is a less consistent feature than splitting of the second sound This splitting is the result of a delay in the tricuspid closure sound Normally, mitral closure slightly precedes tricuspid closure In ASD this is exaggerated The mechanical lag between onset of right ventricular contraction and tricuspid valve closure is increased perhaps in part because of slower rise in ventricular pressure but probably mainly

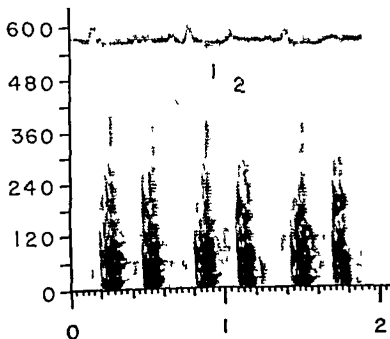


Fig 348 Crescendic construction of S_1 with ASD probably caused by the first sound followed by an early systolic click of pulmonary origin In other cases it may be caused by a mitral closure sound followed by an accentuated tricuspid closure sound Whatever the basis mitral stenosis may be simulated Note split S_2 LLSB in D A (332802)

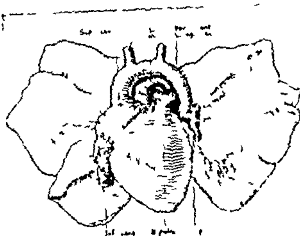


Fig 354 Total anomalous pulmonary venous return Sketch of an anatomic specimen in a patient with figure of eight syndrome All venous drainage from the right lung is into a transverse venous sinus which courses posterior to the heart and joins a persistent left superior vena cava that receives the venous drainage from the left lung Obviously all pulmonary venous return is to the right side of the heart and ASD is by necessity present (Reproduced from MacManus (101) with the permission of the author and publisher)

which appears to be holosystolic because it extends up to the aortic closure sound Often the systolic murmur of ostium primum is whistling in quality (1140)

ANOMALOUS PULMONARY VENOUS RETURN (AVR)

(Syn Anomalous pulmonary venous drainage or connection transposition of the pulmonary veins)

DEFINITION Included in this category is a number of malformations in which at least part of the venous return from the lungs *enters* the right side of the heart rather than the left (1076)

ANATOMIC CONSIDERATIONS There are two *main* categories of anomalous venous return (1) partial (2) total In the latter group the *presence of* an atrial septal defect is essential to survival The varieties of anomalous venous return are illustrated by the classification of cases seen in this hospital and presented in Table 34 (p 393) The figure-of-eight variety is illustrated by the sketch in Figure 354 and the x-ray studies in Figures 355 357 and 358

PHYSIOLOGIC CONSIDERATIONS Physiologically and clinically AVR *particularly* the partial type has many features identical to those of ASD As ASD there is a left to right shunt at the level of the atrium At cardiac catheterization it is often impossible to differentiate the two disorders in both an oxygen step up may be discovered in the right atrium and the catheter may pass into the lung field from the area of the right atrium

CARDIOVASCULAR SOUND Because of the physiologic parallels to ASD all the same changes in the heart sounds are to be expected for example a mid-diastolic murmur at the apex (525 1238 1530) As in ASD because of the left-to-right shunt pulmonary blood flow may be much greater than systemic It is little wonder then that there may be a striking pulmonary systolic murmur

Occasionally in total AVR of the snowman figure-of-eight or cottage loaf variety (so called because of the radiologic appearance created by the left sided superior vena cava and the dilated though normally positioned right *superior vena cava*) a continuous murmur stethoscopically and phonocardiographically identical to that of patent ductus arteriosus may be located under the right clavicle (777) Anomalous (i.e right sided) PDA has been a mistaken diagnosis in certain of these cases (see Fig 359) The site of generation of the murmur is clearly the point where the persistent left superior vena cava enters the normal superior vena cava* As demonstrated in Figure 356 and 357 angiocardiograms show striking dilution at this site The superior vena cava usually has a bulge to the right at this site and pulsates very actively suggesting an arterial structure In fact in roentgen kymograms or electrokymograms the character of the pulsations may appear to be arterial Furthermore at operation exposure of the vena cava reveals a thrill over the superior vena cava and

* Actually the left superior vena cava empties into the innominate vein which in turn joins the right superior vena cava

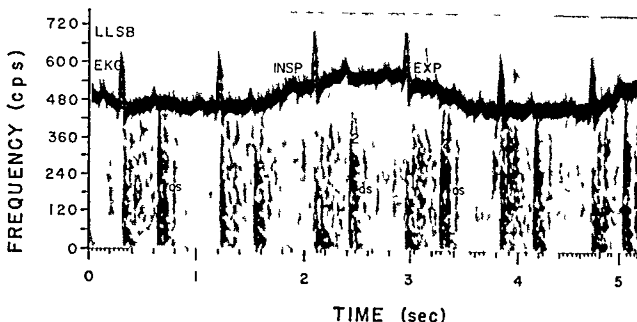


Fig 350 Opening snap in atrial septal defect

A B (792815) a 38 year old woman had a story of indubitable rheumatic fever The clinical signs except for the opening snap were characteristic of atrial septal defect as were also the findings of cardiac catheterization The patient died five days following open heart surgery for closure of the atrial septal defect Necropsy revealed very minimal fibrosis in the aortic leaflet of the mitral valve and associated chordae tendineae

The basis of the opening snap was not clear in this case the mitral valve could not be completely exonerated It should be noticed that the first sound at the apex was normal in quality and in timing

Nadas and Alimurung (1138) found an apical diastolic murmur in 19 of 100 patients with simple ASD Among 20 patients with congenital malformations of the heart and apical diastolic murmurs the diagnosis was ASD in 8 VSD in 5 PDA in 4, Eisenmenger complex in 2 and single ventricle in 1

4 Early systolic click which also occurs commonly with the dilated pulmonary artery of mitral stenosis

5 The occasional occurrence of an AV opening snap of probable tricuspid origin in ASD increases the simulation of mitral stenosis

ENDOCARDIAL CUSHION DEFECTS

(Common atrioventricular canal AV communis persistent common AV canal, ostium primum)

ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS The designation suggested for this general group by Campbell and Missen (228) is used All members of the group result from faulty development and fusion of the dorsal and ventral AV endocardial cushions The result is at one extreme a persistent common AV canal in which there is a single anterior and a single posterior AV cusp, each common to the two ventricles and a single AV orifice continuous with a persistent ostium primum above and a high ventricular septal defect below The other extreme result is persistent ostium primum Although ostium primum defect with normal AV valves is reported (*cf* case 3 of Blount (118)) the usual experience is an associated cleft of the anterior aortic leaflet of the mitral valve (1499) Occasionally the tricuspid valve is also affected Discussion of this group is introduced at this point because clinically atrial septal defect is simulated The shunt is usually from left to right and the patients are acyanotic Defects of this type occur commonly in association with mongoloid idiocy

CARDIOVASCULAR SOUND The fixed splitting of S seen with secundum type of ASD occurs also with the primum type A systolic murmur of mitral regurgitation is common When the other features of ASD are present a loud systolic murmur which is well heard in the left axilla is a valuable clue to identification of the defect as an ostium primum variety One must not be confused by a pulmonary ejection systolic murmur



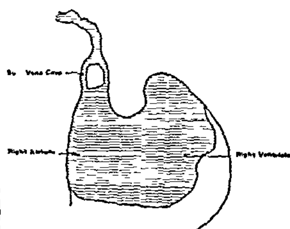


Fig 394 Anomalous pulmonary venous return

Film from an angiocardiographic series illustrating dilution of the live and abrupt widening of the vena cava at the point where the anomalous pulmonary vein enters it A murmur is likely to be generated at this point

great swirls are visible where the fully oxygenated blood returning from the lung meets the dark venous blood (e g K F 600600)

The essential feature of the superior vena caval murmur in total AVR the feature which results in simulation of PDA is its maximum in late systole Pertinent may be the flow meter observation of Brecher (172) and of Nilson and Kramer (1145) that flow in the superior vena cava is maximal during ventricular systole presumably because the atrioventricular diaphragm is pulled down markedly during that phase

Although in some cases the murmur is loudest on the left of the sternum (in these instances it probably arises in the left SVC) and has the fickleness and the humming quality of a venous hum the murmur mentioned above simulates that of PDA precisely except for its location

The murmur of pulmonary regurgitation is described in some cases (192) (1104) I have been surprised by the infrequency of fixed splitting of S_2 in cases of AVR Ongley (1159) makes the same statement Many other authors do not mention splitting of S_2 as a feature of these cases occasionally writers (e g 396) have mentioned it

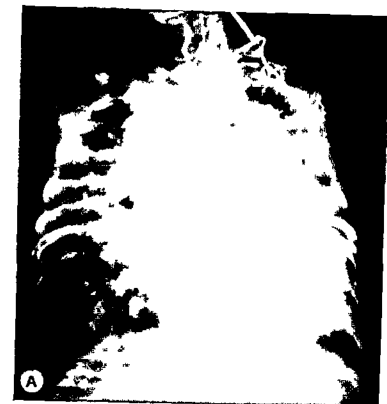
VENTRICULAR SEPTAL DEFECT

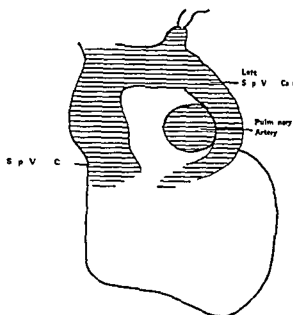
including the Eisenmenger complex and the syndrome of associated aortic regurgitation

(Syn Roger s disease maladie de Roger VSD)

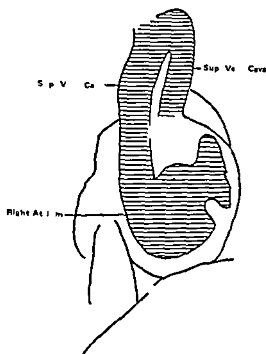
DEFINITION Any communication between the two ventricles via one or more foramina in the ventricular septum

ANATOMIC CONSIDERATIONS (77 1509) The anatomical variability is the impressive feature More involve primarily the membranous septum than the muscular septum However even in the instance of involvement of the membranous septum there is likely to be some involvement of the muscular septum as well There is little basis for the old concept that benign VSD (Roger s disease) is usually muscular or low whereas serious VSD was high or membranous The most frequent variety of defect appears to be one in which the membranous septum is primarily involved the defect lies as far as the left side is concerned below the aortic valve in the portion of the septum that contributes to the outflow tract of









Figs 352(A) and 353(B) Total AVR

PA (352) and lateral (353) views made simultaneously in an angiocardiographic series in a child with the figure of eight syndrome Pulmonary veins demonstrated and superior loop opacified





Fig. 357 (*above*) and 358 (*below*). Total AVR. Ordinary radiograms of the chest in patients with the typical figure of eight syndrome. The left part of the SVC (arrows in Fig. 357) is always the least dense component of the anomalous cardiovascular stripe.

paragraph the intimate anatomical relationship to the aortic valve is evident. It is not surprising that retroversion of the right anterior coronary cusp with aortic regurgitation may occur from lack of proper suspension and support for that cusp.

Occasionally the communication is located above the crista supraventricularis in the outflow tract of the right ventricle. Defects in the muscular septum are likely to be multiple.

Anatomically the Eisenmenger complex is the tetralogy of Fallot minus pulmonary stenosis. It differs from simple VSD in that there is dextroposition or overriding of the aorta. *Physiologically* the Eisenmenger complex has parallels to T. F. As in T. F. there is obstruction to pulmonary blood flow but the obstruction is located at the level of the pulmonary arterioles.

Acquired VSD occurs with myocardial infarction (see p. 425) and after stab wounds of the heart (211B, 477A, 1022A). Nonpenetrating trauma to the chest is occasionally the cause of perforation of the septum (1420).

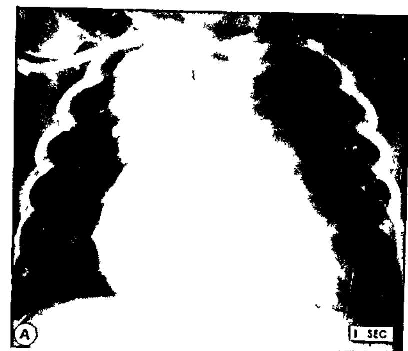
PHYSIOLOGIC CONSIDERATIONS. Because of the greater pressure in the left ventricle than in the right, the shunt in VSD is from left to right under ordinary circumstances. As a result, pulmonary blood flow and mitral orifice flow (but not aortic flow) are likely to be greatly increased. Only during systole is the pressure in the two ventricles different, with the higher pressure in the left ventricle. Therefore, the shunt and murmur produced thereby occur only in systole.

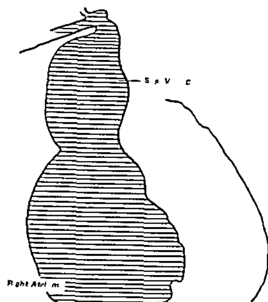
The clinical manifestation and gravity of VSD are determined by the size of the defect and the resistances in the systemic and pulmonary cir-

TABLE 14

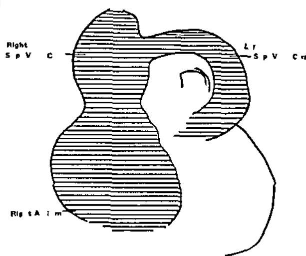
Types of anomalous venous return (1076)

Type	No. of Cases
Partial transposition	
To inferior vena cava	1
To superior vena cava	
Right superior pulmonary vein	3
Left superior pulmonary vein	1
To right atrium directly	1
Left pulmonary vein(s) to right atrium via persistent superior vena cava on left & coronary sinus	–
Total transposition	
To right atrium (unspecified point of entry)	2
To right atrium (via coronary sinus)	1
Figure of eight syndrome (persistent superior vena cava on left to superior vena cava on right)	6
To superior vena cava on right via azygos vein	1









Figs 355(A) and 356(B) Total AVR

Two films from an angiocardiographic series in K. I. (506600) a 7 year old child with continuous PDA like murmur under the *right* clavicle. Fig 355 demonstrates the massive dilatation of the superior vena cava and right atrium. At the time that the pulmonary veins are demonstrated by contrast substance (Fig 356) the left SVC is opacified *and the right SVC and right atrium are re opacified. In the right SVC "swirling" is demonstrated in Fig 356.* At operation the swirling mixture of arterial and venous blood was striking.

the left ventricle, and as far as the right side is concerned, the defect communicates with the right ventricle under and behind the septal leaflet of the tricuspid valve. The stream of blood in such instances must pass among the tricuspid *chordae tendineae*. When bacterial endocarditis occurs in association with VSD the vegetations are often located primarily on these chordae. There are often anomalous tendons traversing the right side of the defect with attachment at the upper and lower margins like the strings of an Aeolian harp (see Fig 186, also, Gould (580) and Taussig and Semans (1462)).

In the variety of VSD described in the last





Figs 35[illegible] (*above*) and 358 (*below*) Total AVR Ordinary radiograms of the chest in patients with the typical figure of eight syndrome. The left part of the SVC (arrows in Fig 35[illegible]) is always the least dense component of the anomalous cardiovascular stripe

paragraph the intimate anatomical relationship to the aortic valve is evident. It is not surprising that retroversion of the right anterior coronary cusp with aortic regurgitation may occur from lack of proper suspension and support for that cusp

Occasionally the communication is located

TABLE 14

Types of anomalous venous return (10 [illegible])

Type	No. of Cases
Partial transposition	
To inferior vena cava	1
To superior vena cava	
Right upper pulmonary vein	3
Left upper pulmonary vein	1
To right atrium directly	1
Left pulmonary vein(s) to right atrium via persistent superior vena cava on left & coronary sinus	[illegible]
Total transposition	
To right atrium (unspecified point of entry)	2
To right atrium (via coronary sinus)	1
Figure of eight syndrome (persistent superior vena cava on left to superior vena cava on right)	8
To superior vena cava on right via azygos vein	1

above the crista supraventricularis in the outflow tract of the right ventricle. Defects in the muscular septum are likely to be multiple

Anatomically the Eisenmenger complex is the tetralogy of Fallot minus pulmonary stenosis. It differs from simple VSD in that there is dextroposition or overriding of the aorta. *Physiologically* the Eisenmenger complex has parallel to T F. As in T F there is obstruction to pulmonary blood flow but the obstruction is located at the level of the pulmonary arterioles

Acquired VSD occurs with myocardial infarction (see p 42[illegible]) and after stab wound of the heart (241B 477A 1022A). Nonpenetrating trauma to the chest is occasionally the cause of perforation of the septum (1120)

PHYSIOLOGIC CONSIDERATIONS Because of the greater pressure in the left ventricle than in the right the shunt in VSD is from left to right under ordinary circumstances. As a result pulmonary blood flow and mitral orifice flow (but not aortic flow) are likely to be greatly increased. Only during systole is the pressure in the two ventricles different with the higher pressure in the left ventricle. Therefore the shunt and murmur produced thereby occur only in systole

The clinical manifestations and gravity of VSD are determined by the size of the defect and the resistances in the systemic and pulmonary cir

Fig 359 Total anomalous venous return of figure of eight type Intense murmur has greatest amplitude and frequency span in the region of the second heart sound (II) as in the murmur of patent ductus arteriosus Recorded under midclavicle in first right interspace

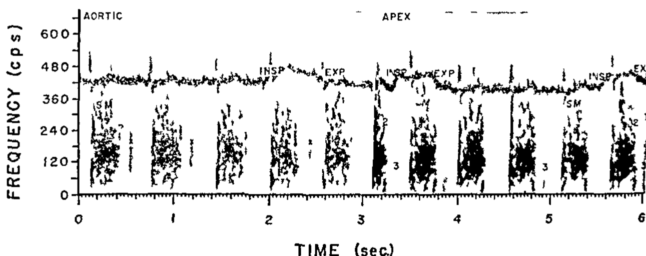


Fig 360 Maladie de Roger

L M (759824) $5\frac{1}{2}$ year old female demonstrated typical cardiac catheterization findings with a pulmonary artery pressure of 41/8 mm Hg In the aortic area as elsewhere there is a holosystolic murmur stopping with the first (aortic) component of S_2 S_2 is minutely split in inspiration A third heart sound gallop is demonstrated at the apex and is probably produced by hyperdynamic filling of the left ventricle The snappy sound marked x seems to occur later than the third heart sound and its nature is unclear See Figure 362 for a similar finding

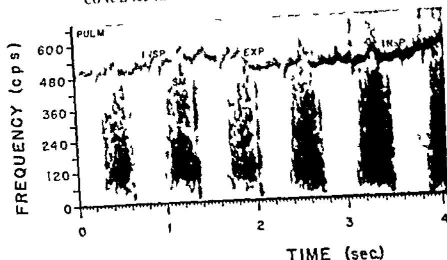


Fig 361 Maladie de Roger

Pulmonary area in M L (V64 4) 19 year old asymptomatic female There was no mid-diastolic rumble at the apex There is a loud holosystolic murmur at the left sternal border In the pulmonary area S_2 is slightly split with moderate accentuation of the second component which is probably pulmonary

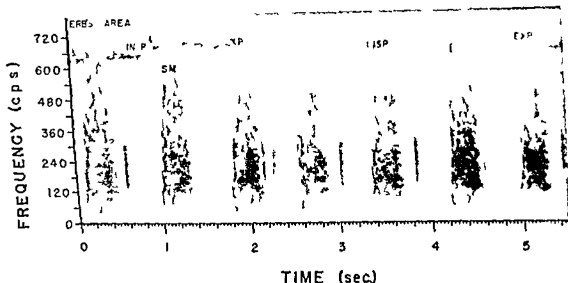


Fig 362 Ventricular septal defect

Third left interspace in T R (51219) 14 year old female The murmur was discovered by the father (as a thrill) when he was holding the infant at the age of two weeks EKG shows balanced axis and partial right bundle branch block The heart is at the upper limit of normal for size with increased prominence of the pulmonary artery which has increased pulsation The murmur extends throughout systole S_2 is very slightly split There is an early diastolic snap mainly in expiration which is not understood See fig 145 149 and 360 for diastolic clicks which like this may be extracardiac in origin

cuits On these factors depend the size and direction of the shunt and the level of pulmonary pressure (1376) Overriding of the aorta is probably only a minor determinant

Whether with small defects—especially of the muscular septum—closure of the defect with systolic contraction of the ventricle occurs can only be speculated Factors which alone or in various combinations may reduce (or almost totally reverse) the right to-left shunt include

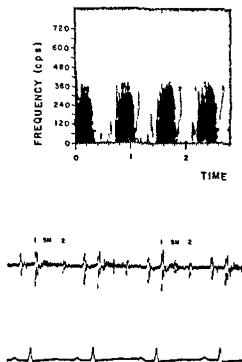


Fig 363 Ventricular septal defect
Above Left sternal border in J T (276556) with VSD Musical element to murmur Presystolic gallop (a) *Below* Oscillographic PCG corresponding to A

(1) increased resistance to pulmonary flow through narrowing of the *pulmonary arterioles or from* pulmonary stenosis and (2) dextroposition (overriding) of the aorta As in the case of ASD, it is thought that two factors, alone or in combination, *may be operative in producing the increased* pulmonary vascular resistance congenital anomaly, i e, persistence of the fetal pattern of the small pulmonary vessels and changes from *the wear and tear of increased flow*

Returning to the left ventricle through the mitral valve is the usual amount of blood swelled by the addition of that volume which is shunted through the defect and recirculated through the lungs The mitral diastolic murmur is produced by this increased volume of blood The dilated left ventricle contributes The stroke volume of the left ventricle is increased and the diastolic volume by necessity increased

In the Eisenmenger complex cyanosis usually *cyanosis tardive* often appearing first in the teens is present because of pulmonary *hypertension* There are certain physiologic similarities to tetralogy of Fallot the obstruction to pulmonary flow

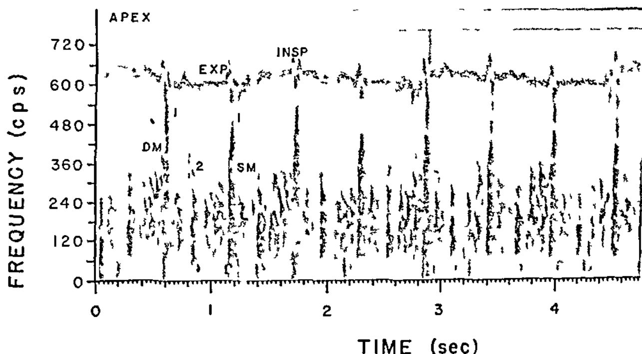
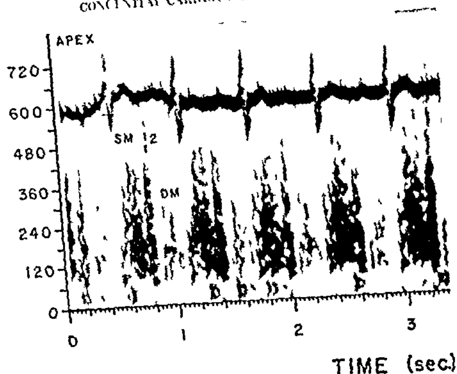
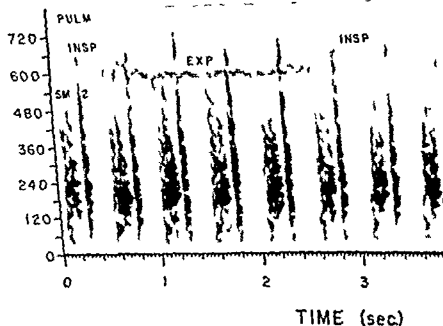


Fig 364 Carey Coombs *murmur with VSD*
L C (B12914) 5½ year old female had sieve like interventricular septum with five perforations There is an impressive diastolic murmur beginning after a gap following S and displaying a presystolic crescendo The mitral valve was normal



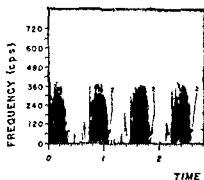
A



B

FIG. 365. VSD with pulmonary hypertension.

S. H. (Ba 09) had SBE at the age of 4 years. At the age of 5 years the diagnosis of VSD (with systolic pulmonary pressure of 80 mm Hg) was established by cardiac catheterization. There is a loud mid diastolic rumble at the apex. The systolic murmur in the pulmonary area is not quite holosystolic as is usually true with this degree of pulmonary hypertension.



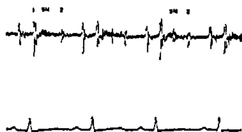


Fig 363 Ventricular septal defect

Above Left sternal border in J T (276556) with VSD Musical element to murmur Presystolic gallop (a) *Below* Oscillographic PCG corresponding to 1

(1) increased resistance to pulmonary flow through narrowing of the pulmonary arterioles or from pulmonary stenosis and (2) dextroposition (overriding) of the aorta As in the case of ASD, it is thought that two factors, alone or in combination, may be operative in producing the increased pulmonary vascular resistance congenital *anomaly, i e, persistence of the fetal pattern of the small pulmonary vessels and changes from* the wear and tear of increased flow

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In the Eisenmenger complex cyanosis usually *cyanosis tardive* often appearing first in the teens is present because of pulmonary hypertension *There are certain physiologic similarities* to tetralogy of Fallot, the obstruction to pulmonary flow

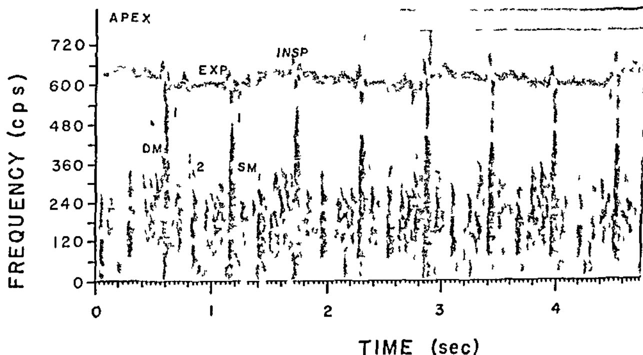


Fig 364 Carey Coombs murmur with VSD

L C (B12914) 5½ year old female had sieve like interventricular septum with five perforations There is an impressive diastolic murmur beginning after a gap following S_2 and displaying a presystolic crescendo The mitral valve was normal

i in the pulmonary arteriolar tree rather than pulmonary valvular area

Gorlin formula for estimating orifice size has been used to estimate the size of ventricular defect (1592)

Some maintain that pulmonary resistance tends to increase progressively in VSD with large shunt On the other hand Wood and his colleagues insist that high pulmonary resistance does not develop slowly over the years but is determined at or shortly after birth As cardiac catheterization ages an answer to this difference of opinion will be forthcoming Truth may be found in both statements A fascinating recent observation (5027 5028) is that of acquired pulmonary stenosis in VSD a phenomenon which is demonstrated by repeat catheterization and accounts for clinical improvement after the first year

CARDIOVASCULAR SOUND The typical Roger murmur is holosystolic intense burying both the first and the second sound maximal at the left sternal border at about the level of the third and fourth interspaces The murmur is characteristically accompanied by a thrill in the same area There may be a musical element to the murmur discernible by ear and SPCG against the noisy background At the apex there may be a Carey Coombs type of blubbering mid-diastolic murmur due to torrential mitral flow

When there is increased resistance to pulmonary flow the systolic murmur tends to become shorter i e limited to the first part of systole and may disappear completely As pulmonary resistance increases the pattern of contraction in the right ventricle comes to resemble that in the pure pulmonary stenosis Peak pressure is attained late in systole Consequently if the pattern of left ventricular systole remains unchanged a pressure gradient across the septal defect will exist only in the first part of systole

There is reason to think that there is often a pulmonary early systolic click and a separate systolic murmur due to dilated pulmonary artery



Fig 68 VSD with aortic or pulmonary regurgitation

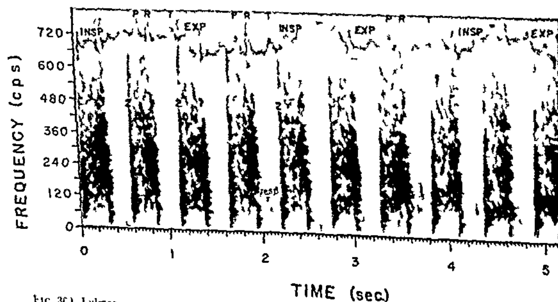


Fig 361 Pulmonary regurgitation in VSD with severe pulmonary hypertension and reversed shunt 11SB in S C M (252896 46 413) 30 year old female has hemialbetosis as well as cyanotic congenital heart dis e Repeatedly nea observer have misinterpreted her murmur as systolic Her rate is always rapid with about equal lengths of systole and diastole and the murmur is not typically decrescendo in fact it becomes louder in late diastole

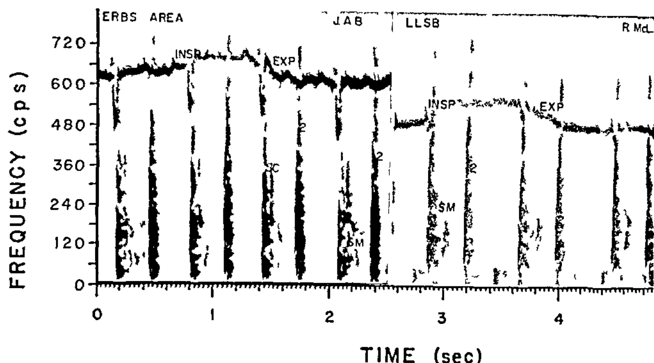


FIG 366 VSD with pulmonary hypertension

J A B (756952) 10 year old female had the tentative diagnosis of ASD Cardiac catheterization showed left right shunt at the ventricular level and marked pulmonary hypertension (122/73 mm Hg) The absence of splitting of S_2 would tend to exclude ASD without catheterization The early systolic click and absence of systolic murmur *are consistent with VSD with pulmonary hypertension or with pulm hypertension alone LLSB in R McL* (62593) 25 year old male with VSD proved by cardiac catheterization which also showed the pulmonary artery pressure to be 140/68 mm Hg The recordings in other areas were essentially the same as shown here Of note are (1) the accentuated S (2) short unimpressive early systolic murmur suggesting that of high pulmonary artery flow and/or pulmonary artery dilatation

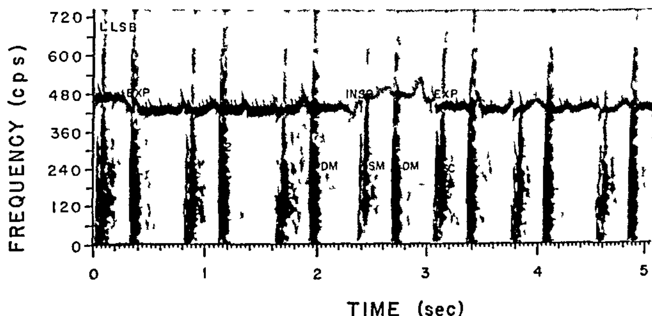
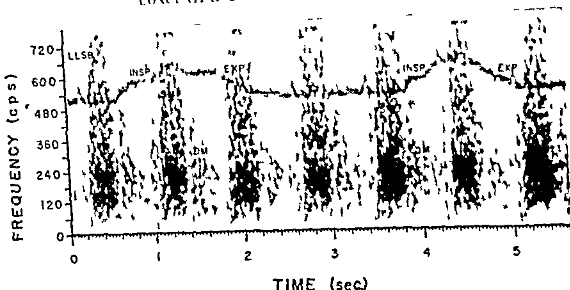


FIG 367 VSD with pulmonary hypertension and pulmonary regurgitation

D M S (760418) 13 year old female had findings of cardiac catheterization consistent with VSD pulmonary artery pressure was 100/48 mm Hg In this recording from the left sternal border note the loud S_2 followed by decrescendo early diastolic murmur and the early systolic click followed by a murmur which has the character of an ejection systolic murmur and may *in fact be produced by the right ventricular outflow* rather than flow through the VSD Systemic diastolic pressure was normal



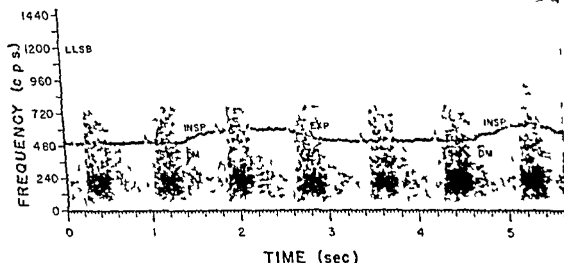


Fig. 371. VSD with aortic regurgitation.

E. R. (266 11), 26 year old white female was thought to have patent ductus arteriosus but none was found at operation. On resurvey it was concluded that the oxygen step up on cardiac catheterization probably occurred in the outflow tract of the right ventricle rather than in the pulmonary artery.

systole it may be completely impossible to differentiate VSD and AR or PR from AS and AR.

A diastolic murmur of pulmonary regurgitation (e.g., Fig. 367) may occur. It was described in four of 19 cases of VSD in infants and children of ages from 3 months to 14 years (11). In some cases of VSD with pulmonary hypertension it is extraordinarily loud (see Fig. 369).

In the Eisenmenger complex there is usually little or no murmur attributable to the VSD. There is however likely to be a pulmonary early systolic click followed by decrescendo systolic murmur caused by dilated pulmonary artery and pulmonary hypertension.

The second sound with VSD, particularly of the Roger type, may be split to a moderate degree (Fig. 361). The mechanism of the splitting is probably the same as that in mitral regurgitation. The aortic valve closes before the pulmonic

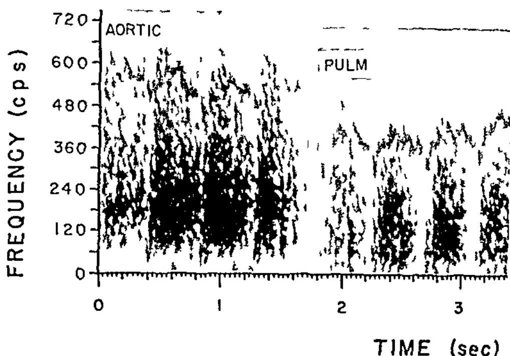


Fig 370 VSD with aortic regurgitation

Aortic and pulmonary areas in L S (A88576) 6 year old girl who was explored for patent ductus arteriosus but none was found Cardiac catheterization findings were those of VSD The blood pressure was 160/50 in the arms 200/60 mm Hg in the leg

and high pulmonary blood flow. More often than not, however, these are concealed by the intense murmur generated at the defect(s). A pulmonary *diastolic murmur may result from dilatation of* the pulmonary artery, especially if pulmonary hypertension supervenes.

Most of the cases in whom closure of VSD by open heart techniques was deemed advisable have had a non holosystolic murmur, an apical diastolic rumble and some degree of pulmonary hypertension. In fact, the incidence of the Carey Coombs murmur is much greater in such cases than in the "classical case" of *maladie de Roger* with holosystolic murmur. Despite increase in pulmonary artery pressure these patients must have high flow through the defect. Indeed, the high flow is probably responsible in large part, for the increase in pressure, there being little increase in pulmonary resistance. As far as reducing the length of the systolic murmur is concerned, it probably matters little whether the pulmonary hypertension *is the effect of high flow* or high pulmonary resistance.

Gasul and his colleagues (527, 528) have observed that a number of *patients with VSD who* previously had a loud, booming second sound in *the pulmonary area later had a definitely diminished* P_2. Recatheterization demonstrated the phenomenon of acquired pulmonary stenosis *in most of these patients. Hypertrophy in the* infundibular area of the right ventricle had apparently developed and created a resistance to *outflow from right ventricle to pulmonary artery*.

The syndrome of VSD and aortic regurgitation (Fig 370) (*31, 68, 329, 735, 854, 1115, 1137* (p 328), 1207, 1462 and 1592) can be mistaken for patent ductus arteriosus. It may also simulate the *combination of aortic stenosis and regurgitation*. Graphic differentiation is possible when the systolic murmur generated at the VSD is holosystolic and the *decrescendo murmur of aortic* regurgitation begins immediately with the second heart sound; a continuous murmur is created *rather than the double murmur (with interruption* after the systolic murmur) as in AS and AR. Furthermore, by graphic means the shape of the *continuous murmur of VSD AR* is more likely to be as represented in Figure 368 than like that typical of PDA (Fig 415). The same confusion *may be created by VSD* with pulmonary regurgitation. When the systolic murmur is not holo

(M.B. 205877) Serious cardiac dysrhythmias sometimes occur (270, 1393). About a third of involved persons are mongoloid idiots. The diagnosis has been made in life by angiocardiograms which at the time of opacification of the left ventricle may show the aneurysm. Occasionally there is rupture of the aneurysm and development of the typical clinical and physiologic features of ventricular septal defect.

ANEURYSM OF THE SINUSES OF VALSALVA*

Ventricular septal defect, aneurysm of the ventricular septum and aneurysm of the coronary sinus have a common denominator: all are congenital malformations of the aorto-ventricular septum (413). Sometimes aneurysm of a sinus of Valsalva is associated with one of the other two lesions (193, 411, 678, 796). Syphilis can also cause such aneurysms; they may occur with subacute bacterial endocarditis and with the Marfan syndrome, and some cases have their basis in idiopathic cystic medial necrosis.

The right anterior coronary sinus is most often affected. Unruptured aneurysm may be associated with a nonspecific systolic murmur and with the diastolic murmur of aortic regurgitation (1439). With rupture of the aneurysm into the right atrium, right ventricle or pulmonary artery, the patient abruptly develops a continuous murmur with the plateau-decrescendo pattern shown in Figure 368. Graphically this continuous murmur is distinguishable by its pattern from the Christmas-tree-decrescendo double murmur of AS-AR, but with more difficulty from the zigzag continuous murmur of PDA. It is difficult or impossible to distinguish it, however, from the murmur of VSD with AR; other clinical features would be relied on. The murmur of ruptured aortic sinus is often loudest to the right of the sternum or over the mid and lower sternum—a feature which aids differentiation from the murmur of PDA.

PURE PULMONARY STENOSIS (PPS)

(Syn. Pure pulmonary stenosis, valvular and infundibular pulmonary stenosis with intact ventricular septum)

ANATOMIC CONSIDERATIONS. There are two main anatomical varieties of pulmonary stenosis—valvular and infundibular. In addition an exceedingly rare supravalvular type is described (see below). Pure pulmonary stenosis is most often of the valvular type. Rarely both types are present in the same patient. Among 33 patients Levinson and Weinman (848) concluded that valvular stenosis was present in 29, infundibular stenosis in 3, and both varieties in 1. The valve cusps are fused together in a dome-shaped diaphragm with a central perforation. The size of the orifice may be reduced to a fraction of a square centimeter. In the infundibular variety of pulmonary stenosis the ridge-like obstructing element is fibrous and muscular in nature and located in the vicinity of the crista supraventricularis. Between the obstructing ridge and the normal pulmonary valve there is the so-called infundibular chamber ("third ventricle" of Kjellberg) which is walled by ventricular myocardium. Post-stenotic dilatation usually occurs in striking fashion in the pulmonary artery, but probably only with the valvular form of the disease. Brock (179B) has written a monograph on the anatomy of congenital pulmonary stenosis.

PHYSIOLOGIC CONSIDERATIONS. The essential feature of pulmonary stenosis is an appreciable pressure gradient across the pulmonary valve (or infundibular zone). Elevation of right ventricular pressure and depression of pulmonary artery pressure are present. Peak systolic pressure in the right ventricle may exceed 200 mm. Hg in extreme instances. Pressure loss across the valve is accounted for on the basis of the increased velocity of the blood at the stenosis and the large increase in kinetic energy as opposed to lateral pressure energy.

So-called relative pulmonary stenosis results from increased pulmonary blood flow, as in ASD, AVP and VSD (see these entities). In total AVR of the ... type (287) a difference in systolic pressure across the pulmonary valve of as much as 42 mm. Hg may occur on the basis of high flow alone. Randolph, Nadas and Goodale (1320) found a 34 mm. gradient in a case of ASD. Interestingly, Contro, Miller and Derrick (287) could find no correlation between the volume of flow and the pressure gradient. This result suggested to them that another factor, presumably dilatation of the right ventricle and pulmonary artery, is operative.

*See reference 411.

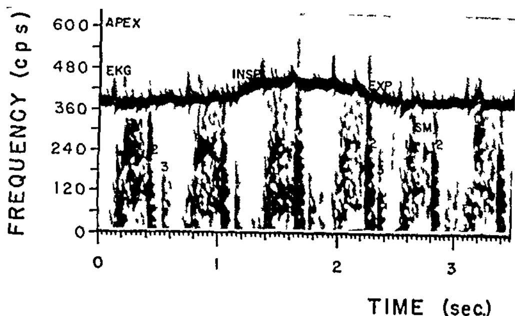


Fig 372

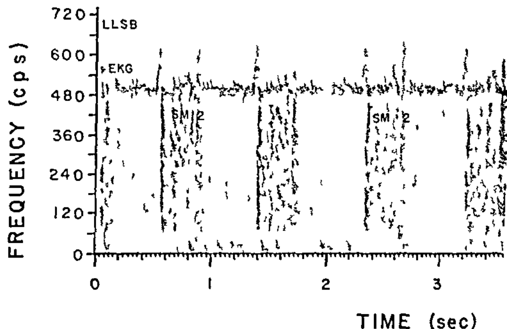


Fig 373

Figs 372 and 373 Differentiation of MR and VSD

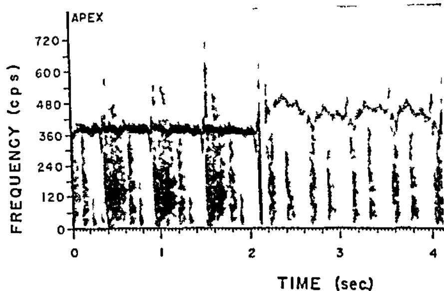
A B (735323) 16 year old female was treated for SBE due to *Streptococcus faecalis* Whether the underlying lesion was VSD or MR was not clear At times the systolic murmur seemed loudest at the apex with third heart sound and possibly a short diastolic rumble At other times the systolic murmur seemed loudest at the left sternal border The SPCG is consistent with either diagnosis since both could account for holosystolic murmur S_3 and short diastolic murmur The third sound gallop is of left ventricular origin it maintains a constant relationship to the first (aortic) component of S

probably because the left ventricle has two avenues of ejection

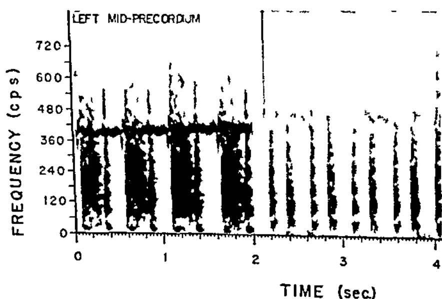
ANEURYSM OF THE MEMBRANOUS SEPTUM

Weakness may result in ballooning to the right of part or all of the membranous septum, usually into the outflow tract of the right ventricle A systolic murmur maximal at the left sternal border and suggesting ventricular septal defect or pulmonary stenosis, is present in some cases, absent in others (1438) The aneurysm may produce enough obstruction to right ventricular outflow to result in right sided heart failure

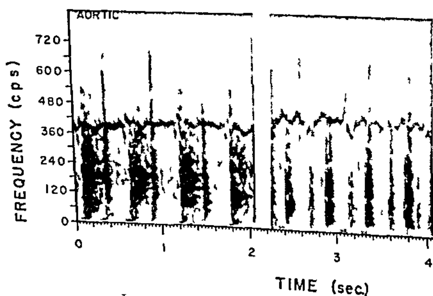
[5] See reference 231



C



D



E

FIG 3 4 C D E See legend Figure 374 1

Anatomically, in cases of valvular PPS, it is difficult to imagine closure of the pulmonary valve in the usual sense However, there appears to be a to and fro snapping of the diaphragm a doming upward with ventricular systole and a snapping back toward the ventricle when pressure there falls below that in the pulmonary artery Furthermore, pulmonary regurgitation of

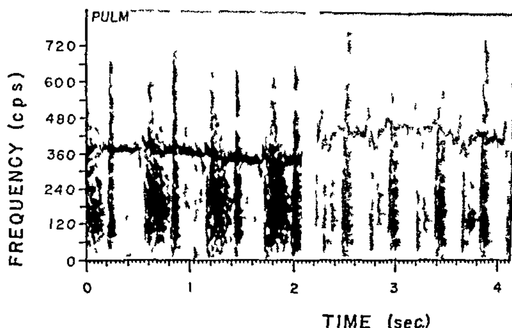


Fig 374 A Surgical closure of VSD

Recordings before (*left*) and after (*right*) operation on K I (742684) 8 year old patient in whom a large (12 by 18 mm) defect high in the right ventricular outflow tract distal to the crista supraventricularis was closed surgically Pulmonary artery pressure was 65 mm Hg

Before operation there was a decrescendo systolic murmur which stopped shortly before S_2—typical of the murmur in VSD with moderate pulmonary hypertension In the pulmonary area there was an early systolic click distinct from S_1 introducing the murmur There was at the apex a protodiastolic gallop caused by hypervolemic filling

After operation S_2 was slightly split (right bundle branch block had appeared) the systolic murmur and gallop were gone the early systolic click was seen unconcealed by murmur (see LLSB) and there were systolic clicks probably of pericardial origin

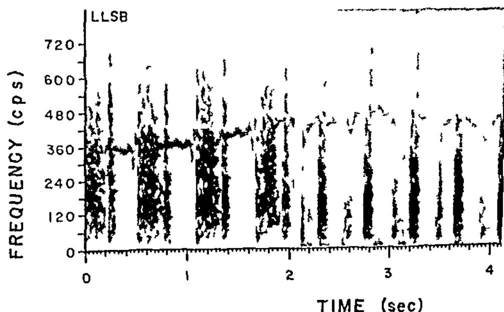


Fig 374 B See legend Figure 374 A

chamber in which the pressure curve has the contour of that in the ventricle but has the same low systolic peak as that in the pulmonary artery. A simulating pattern which can be confused is produced if the catheter tip intermittently flips through a valvular stenosis with each systole.

Rodbard and Shaffer claim (1300) that there are two varieties of infundibular stenosis: in one the stenosing ridge is largely fibrous and the degree of stenosis is unalterable; in the other type the myocardium constitutes a large portion of the obstructing ridge and through active contraction of this muscle the degree of stenosis is increased in the later part of systole. Engle and her colleagues (428) present convincing evidence that in cases of pure valvular stenosis myocardial hypertrophy in the area of the infundibulum produces a dynamic stenosis in systole. This hypertrophy accounts for the failure of right ventricular pressure to fall to normal immediately after operation. There tends, however, to be a slow fall in right ventricular pressure to normal later after operation.

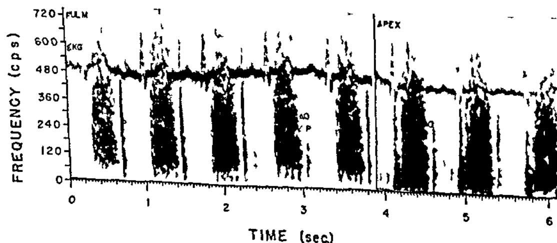
Cyanosis does not occur with IPS unless there is associated ASD. The atrial defect may be only probe (or flap) patency of the foramen ovale with widening of the opening through increase in right atrial pressure.

Pulmonary stenosis is a state of systolic overload of the ventricle. The electrocardiogram shows the characteristic changes, i.e., marked right axis deviation and R waves of large amplitude in the leads at the right end of the precordial series, bespeaking concentric hypertrophy of the ventricle.

CARDIOVASCULAR SOUND. The characteristic *sys*



A



B

Fig. 3?. Pulmonary area (B) in J. W. (158150) with pulmonary stenosis (infundibular type) and ASD. X-ray (A) shows extra bulge on left hand border interpreted as dilated infundibular chamber— so-called third ventricle of Kjellberg (*upper arrow*) and elevation of the apex (*lower arrow*). The murmur can be accounted for entirely on the basis of the pulmonary stenosis, although it is possible that the ASD is contributing. The unusually loud but delayed pulmonary closure sound is consistent with infundibular stenosis. The spectral pattern of the sound is such, however, as to suggest that the valve is not completely normal.

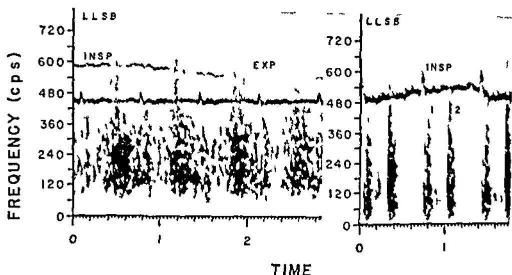


Fig 375 Ruptured sinus of Valsalva occurred in D W (706005) 33 year old woman early in the puerperium The communication was between the posterior sinus and both the right atrium and right ventricle at the level of the tricuspid ring The fistula was closed surgically No diastolic murmur persisted after operation (*right*)

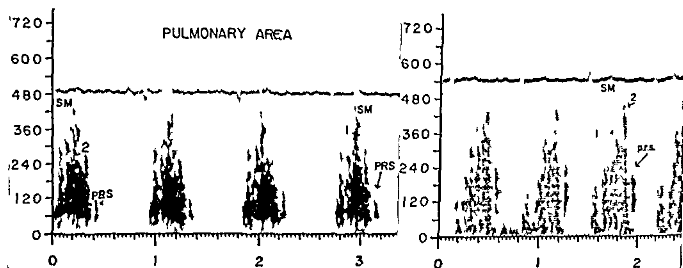


Fig 376 Valvular pulmonary stenosis

In each case the murmur extends to the aortic component of the second sound and there is a delayed relatively diminutive pulmonary component which might be called pulmonary reversal snap (p r s) This has the spectral characteristics of a snap in that its frequency bottom does not extend to zero

significant proportions is unlikely to occur because of the low pressure in the pulmonary artery The ventricleward snap of the stenotic diaphragm—which will be referred to as valve closure—occurs belatedly One might guess that prolongation of the contraction of the right ventricle and the large discrepancy between right ventricular and pulmonary artery pressures both contribute to the delay in pulmonary valve closure Leatham (see below) finds a close correlation between the level of right ventricular pressure and the degree of delay in pulmonary closure as indicated by the pulmonary closure sound

Silber and co workers (1390) suggested a "stenotic index" for semi quantitative estimation of the severity of pulmonary stenosis

Infundibular stenosis can be differentiated from valvular pulmonary stenosis by the "pull back" pressure curves obtained on withdrawal of the catheter from the pulmonary artery during cardiac catheterization With infundibular stenosis pressure is recorded from an intermediate infundibular

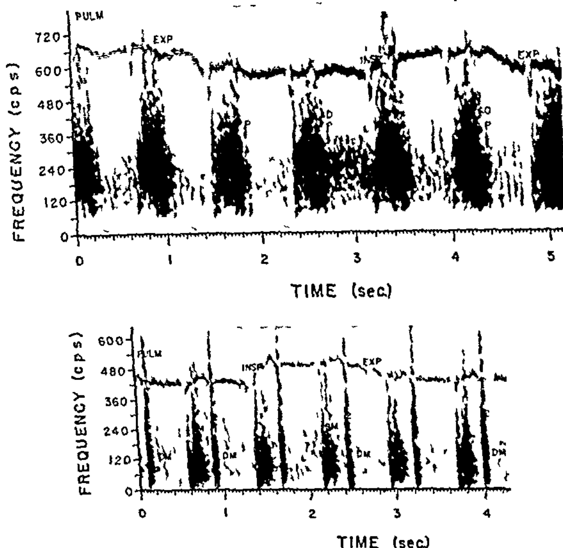


Fig 3 9 Accentuation of PPS murmur with inspiration

L V (504491 1a year old white male demonstrated classical features of PPS—right ventricular hypertrophy post notic dilatation systolic thrill at the left sternal border (Above) Before operation the systolic murmur was strikingly accentuated by inspiration The pulmonary stenosis valvular in type was relieved under direct vision (Below) Same case after operation Pulmonary area The murmur has been converted into an early systolic ejection type resulting mainly perhaps from residual dilatation of the pulmonary artery No splitting of S_2 was seen at any area There is a faint early diastolic murmur

monary artery dilated in connection with the post stenotic phenomenon It is possible that both types of snaps occur Kjellberg et al (800) noted as did Leatham and Vogelpoel (865) that the early systolic click occurred only in mild case Leatham and Weitzman (866) found what they considered to be a typical pulmonary ejection sound in all of 11 patients with very mild pulmonary stenosis i e cases with a normal electrocardiogram and a right ventricular pressure of 40 mm Hg or less The sound in PPS was earlier (av 0 05 sec after S_1) than in cases of pulmonary hypertension with dilated pulmonary artery (av 0 07 sec) However in 33 cases of a more severe variety only five showed the sound and these had right ventricular pressures among the lowest values for the group In one patient the sound appeared after valvotomy

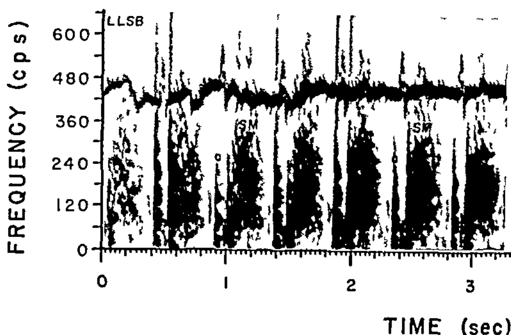


Fig 378 Presystolic gallop with pulmonary stenosis
LLSB in J S (652904) Six year old female with pure pulmonary stenosis of combined infundibular and valvular types Congenital ptosis was an associated anomaly The atrial sound is the loudest sound in this area A diminutive *sound just before the atrial gallop is probably a much delayed pulmonary closure sound*

tolic murmur of pulmonary stenosis is an ejection stenosis murmur like that of aortic stenosis *In* the oscillographic PCG it is diamond shaped, in the SPCG it has the configuration of a Christmas *tree In general, the peak of intensity and fre*quency of this murmur is later after the first heart sound (or QRS) than in the case of the murmur *of aortic stenosis However, as pointed out by* Leatham (862) just as in aortic stenosis in which a silent gap separates the end of the murmur from *the aortic closure sound there is in pulmonary* stenosis a gap between the end of the murmur and the delayed pulmonary closure sound even *though the murmur usually extends up to the* aortic closure sound

Vogelpoel and Shrire (1495) showed that it is *possible to differentiate pulmonary stenosis with* intact ventricular septum from cases with VSD as in tetralogy of Fallot, in the latter case the murmur usually *stops before the aortic closure* sound In the former case as just stated the murmur extends at least to—and usually beyond—*the aortic closure sound The differentia*tion is particularly useful in connection with pulmonary stenosis and ASD with reversed shunt, which may *simulate tetralogy* of Fallot In the experience of Leatham and Weitzman (866) however, *only 7 of 26 cases of tetralogy showed a* murmur which stopped before the aortic closure sound It can at least be stated that when the *murmur does display this pattern, tetralogy can* be diagnosed

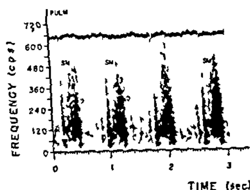
The murmur of PPS is maximal in the pul*monary area It may radiate toward the left* shoulder and to the back, but usually not to the base of the neck on the right as does the murmur of aortic stenosis, *although when very loud it may* even be heard in that area In valvular stenosis the murmur and thrill are likely to be detectable *in the suprasternal notch and maximal in the* second left interspace In infundibular stenosis the murmur is often maximal somewhat lower—*in the third interspace*

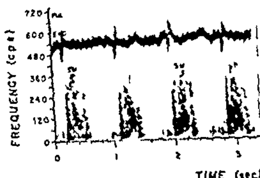
The murmur is likely to be introduced by a snap (described first by Petit (1204)) which is too *late in relation to the QRS to be first heart* sound[7] and is interpreted as being caused by the abrupt doming of the stenotic valve diaphragm *into the pulmonary artery* Another possible mechanism is snapping of the wall of the pul

[7] It may be this snap which has led some (12[illegible]) to conclude that the first sound is accentuated in PPS

with the *production of slight paradoxical splitting* of S in extreme instances. However, the prolongation of left ventricular systole never attain the marked degree seen with pulmonary stenosis. The left ventricle by design a more satisfactory pressure pump is better able to cope with the increased burden. The right ventricle by design primarily a volume pump (1323) shows a quantitatively different reaction to outflow obstruction.

Prolongation of right ventricular systole cannot be attributed (866) to delay in the onset of right ventricular systole since this averaged 0 07 sec from the beginning of the Q wave a figure agreeing with the finding of Coblentz et al (276) Nor is there any abbreviation of left ventricular systole which averaged 0 31 sec a normal value In cases of pulmonary stenosis right ventricular systole averaged 0 39 sec (range = 0 36 0 41 sec)





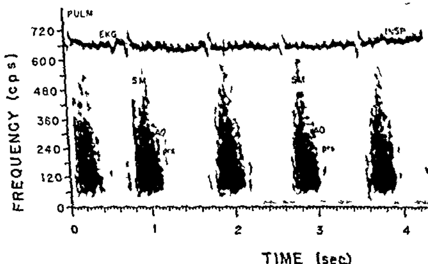


Fig 381 Valvular pulmonary stenosis with ASD

Transventricular valvulectomy was performed soon *after the recording* shown (*above left*) The patient is one of the ... per cent which has no pulmonary reversal sound (Most of the sound *in dia*stole is thought to be of non cardio vascular origin) At operation the anatomy seemed to be typical of valvular stenosis The dome like stenosis would ... The right ventricular muscle was very thick After the valvulectomy pressure in the right ventricle was still between 80 and 100 mm Hg systolic In the recording taken 4 *months after* operation (*bottom*) there is still a ... systolic which however has an earlier peak A delayed pulmonary reversal sound (p r) is now present 18 months after operation the murmur (*above right*) had greatly diminished

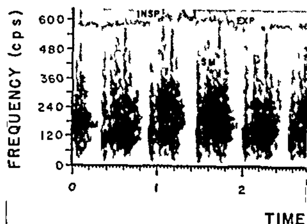


FIG 380 Pulmonary stenosis and regurgitation Pulmonary area in N T (735272) asymptomatic 13 year old female Cardiac catheterization showed a right ventricular pressure of 40/5 mm Hg and mean pulmonary artery pressure of 8 mm The main pulmonary artery was dilated and pulsatile EKG showed slight RAD and RSR pattern in V_1 The split S characteristic of pulmonary stenosis is present The pulmonary component is less delayed or muffled than in more marked cases The diastolic murmur is probably lower pitched than is the case with aortic lesions

There may be an appreciable delay (Fig 377) between the first sound and the onset of the murmur (706, p 143) Mannheimer (1033) suggests that ballooning of an infundibular chamber in valvular PPS may be a factor in this delay He proposes the presence of the S_1 murmur gap as a point differentiating valvular from infundibular PPS He (1035) thought that the peak of the murmur of infundibular stenosis occurs earlier Kjellberg *et al* (800) thought that there are other features which permit differentiation in infundibular stenosis the murmur usually extends only as far as the aortic closure sound whereas in valvular stenosis it is likely to extend beyond it because of the late contracting infundibulum Vogelpoel and Shrire (1495) also thought differentiation was possible However Leatham and Weitzman (866) concluded that there are no differentiating points

Frequently, after operation for pure pulmonary stenosis of the valvular type a considerable systolic murmur persists despite the fact that the operative procedure was thought to have been technically satisfactory The explanation is probably provided by the observations of Engle and colleagues (428) in such cases there is often hypertrophy in the infundibular area of the right ventricle and a dynamic type of outflow obstruction After several months this factor disappears Figure 381 presents the records of a patient with pure pulmonary stenosis who still had a considerable murmur 4 months after operation but had lost it almost completely 14 months after operation

Unlike that of true pulmonary stenosis, the murmur of relative pulmonary stenosis is limited to early systole In organic pulmonary stenosis the delay in the peak of the systolic murmur results from a change in the pattern of right ventricular contraction

The pulmonary closure sound, perhaps better termed pulmonary reversal snap (1082), is always diminutive (except in the mildest cases) and is delayed Its intensity is, however, a poor index of the severity of the pulmonary stenosis, just as the degree of depression of pulmonary arterial pressure is a poor index Variability in the anatomy of the malformed valve, in the proximity of the pulmonary artery to the chest wall, in the thickness of the chest wall and possibly in other factors is too great to permit assignment of quantitative significance to the intensity of the pulmonary reversal sound On the other hand the interval between the aortic and pulmonary closure sounds has been found to bear a fairly direct relationship to the degree of elevation of right ventricular pressure (Fig 382) This relationship no longer obtains when patent ductus arteriosus or VSD is associated However the association of ASD does not vitiate the relationship

Although there is close correlation between right ventricular pressure and the interval between the aortic and pulmonary components of S it is not necessarily correct to assume that the delay of the pulmonary component is caused merely by the fact that a longer time is required for the high pressure in the right ventricle to fall below that in the pulmonary artery It is likely that the pattern of right ventricular contraction is seriously altered with prolongation of systole as an important feature The peak of the systolic murmur is later in systole than is the case with the murmur of aortic stenosis The aortic closure sound may be somewhat delayed in aortic stenosis,

Although the systolic murmur of pulmonary stenosis is reduced in duration and intensity after operation it is never abolished completely even by direct valvotomy. Following operation for valvular pulmonary stenosis the systolic murmur may subside slowly presumably because an element of infundibular stenosis is on the basis of myocardial hypertrophy is reversible.

Atrial gallop occurs frequently (3 109) with pulmonary stenosis as with other instances of systolic overload of the ventricle. Laubry and Pezzi (848) and Blackford and Parker (109) emphasized its audibility over the cervical veins. However Leatham and Weitzman (866) found an atrial sound in only 2 of 33 cases of moderate and severe cases of P.S. An auricular systolic murmur of low intensity occurred in three patients who displayed a prominent *a* wave in the venous pulse of the neck. They (866) speculated with some evidence in support that with atrial systole blood might actually be forced through the stenotic area. Relative tricuspid stenosis is an alternative explanation.

TETRALOGY OF FALLOT (T/F)

(Syn. Triad of Fallot)

DEFINITION. The complex of VSD, PS and overriding or dextroposed aorta constitutes the triad of Fallot. Hypertrophy of the right ventricle completed Fallot's tetralogy but obviously is not a congenital malformation in the sense that the others are but rather a postnatal development in compensation for the malformation.

The designations *trilogy of Fallot* and *pentad of Fallot* are used especially by Fallot's countrymen. The purist raises legitimate objections to these eponyms. Trilogy of Fallot is pure pulmonary stenosis with patent interatrial septum. The simulation of T/F is discussed below. Pentad of Fallot is T/F plus patent interatrial septum. Clinically patients with this combination behave differently from patients with pure T/F.

ANATOMIC CONSIDERATIONS (198 749 1509). The obstruction to normal right ventricular outflow is usually infundibular pulmonary stenosis and less often valvular pulmonary stenosis, a combination of these two, or pulmonary atresia. Older patients coming to autopsy tend to have valvular stenosis which is more severe than the infundibular stenosis (40). In younger patients infundibular stenosis predominates. Most valvular cases have infundibular stenosis of some degree as well.

The pulmonary valve beyond an infundibular stenosis is frequently bicuspid in this syndrome. The bronchial arteries become dilated and assist although inadequately pulmonary blood flow. Poststenotic dilatation of the pulmonary artery occurs only in a minority of the cases as might be expected because in most infundibular stenosis dominates. Usually there is by x-ray a concavity in the region of the pulmonary artery and the cardiac apex is tilted up through enlargement of the right ventricle and possibly shrinking of the left. The net result is a sabot shaped cardiovascular silhouette. Occasionally the pulmonary valve is totally absent, the pulmonary stenosis is infundibular in type and an arterial diastolic murmur is added to the other auscultatory signs of T/F.

The aorta rather than describing an arc to the right and then to the left in its course in the antero-posterior direction tends to pass straighter cephalo-caudad in the sagittal plane. Pattinson and Emanuel (1185) have emphasized enlargement of the ascending aorta in T/F. The aorta narrows fairly abruptly in the vicinity of the isthmus.

The dilated bronchial collaterals which may carry more blood to the lungs than the normal pathways or may be the only route for pulmonary blood flow in cases of pulmonary atresia have been beautifully demonstrated by Hales and Liebow (621). They may be so striking as to distort the esophagus and be otherwise demonstrable on radiologic study (290 291). Even notching of the ribs may occur through dilatation of the intercostal arteries as part of the collateral circulation.

PHYSIOLOGIC CONSIDERATIONS. The basic physiologic defects in Fallot's syndrome are (1) obstruction to flow of blood to the lungs and (2) right to left shunt. The cyanosis, polycythemia and clubbing are well accounted for on this basis. Because of the overriding aorta there is less obstruction to ejection of the right ventricle into the systemic circulation than there is in simple VSD. The pressure in the right ventricle is elevated and at times the right ventricle becomes essentially the systemic ventricle.

Squatting as has been emphasized by Tau

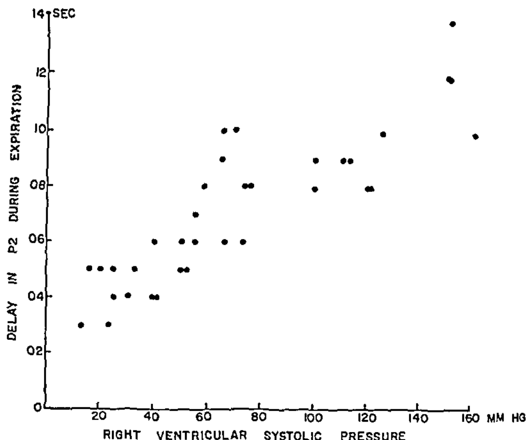


FIG 382 The relationship between right ventricular pressure and the interval separating the aortic closure sound from the pulmonary reversal snap (From Leatham and Weitzman (866))

Phonocardiographically the delayed pulmonary closure sound (or "pulmonary reversal snap") is demonstrable in the overwhelming majority of cases—about 85 per cent of cases of pure pulmonary stenosis (866) When not demonstrable, unusually severe pulmonary stenosis is likely to be present The sound may appear after valvotomy in such cases and if previously present moves closer to the aortic component after operation Stethoscopically Barritt (59) found a "split second sound" in 11 of 33 cases Abrahams and Wood (3) described a split P_2 in 12 of 19 cases of mild stenosis, but in all severe and moderate cases P_2 was unitary and of presumed aortic origin In regard to the pulmonary closure sound there seems to be no difference between valvular and infundibular stenosis

Kjellberg *et al* (800) described a bizarre case in which the obstructing lesion was a diaphragm with central perforation located in the first portion of the pulmonary artery with a normal pulmonary valve and infundibulum below In this case, three sounds were present in the general vicinity *of the normal second heart sound* These were interpreted as aortic closure sound pulmonary closure sound and reversal snap of the anomalous diaphragm Williams Lange and Hecht (1564) have observations on cases of supravalvular pulmonary stenosis

Pulmonary regurgitation (Fig 380) occurs rarely in congenital pulmonary stenosis Leatham and Weitzman (866) found it in one case of valvular stenosis The low pulmonary artery pressure probably militates against such a murmur It is likewise rare for pulmonary regurgitation to develop after valvotomy although such occasionally is observed It developed in 8 of 22 patients in whom open valvuloplasty was performed by Swan plus all three of the patients in whom a segment of the valve was excised (120) It has also been encountered after transventricular valvulotomy (226) Pulmonary regurgitation is however a benign lesion (42, 476) when pulmonary hypertension is not present

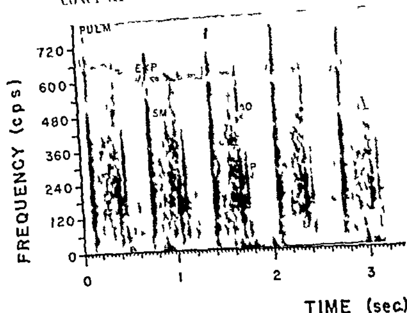


Fig. 385 A. Pulmonary stenosis with ASD.
C. Y. (?75391) 12 year old female had a clinical picture consistent with T/F. The sounds are more consistent with PS (with ASD also), a diagnosis confirmed by cardiac catheterization. Contrast with characteristic pattern of T/F in Figure 384 and last half of 385 B.

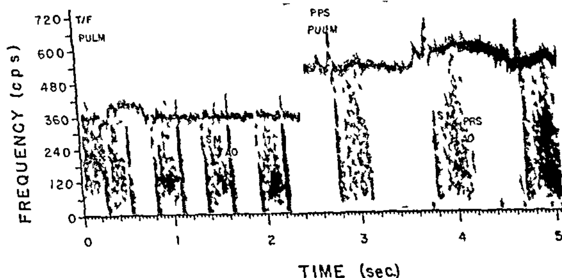


Fig. 385 B. Tetralogy of Fallot compared with pure pulmonary stenosis.
On left, pulmonary stenosis in L. K. (?88233) 3½ year old girl with typical clinical, physical, x-ray and surgical findings of T/F. SM is characteristically unitary and in most cycles the systolic murmur stops slightly before A. On right, for comparison, pure pulmonary stenosis.

intensity of the systolic murmur occurs after exercise, especially after exercise with standing—situation in which flow across the stenotic pulmonary valve is reduced and flow through the VSD increased. A rare but physiologically interesting situation which is the converse of T/F and exercise is T/F combined with coarctation of the aorta. In such cases the systolic murmur of T/F is louder and longer and a pulmonary closure sound may be present. The increased systemic

sig (1460), a characteristic posture which patients with tetralogy of Fallot, and less commonly with other varieties of cyanotic congenital heart disease, assume after exertion. The child begins to display the phenomenon when he starts to walk. Social pressures may lead to its disappearance at the age of eight to ten years. Thereafter, crossing the legs with entwining and squeezing may take the place of squatting. Arterial oxygen saturation falls during and after exercise but recovery is more rapid with squatting (Fig. 386). Fall in systemic resistance (996) through peripheral vasodilatation with constant resistance to pulmonary blood flow results in fall in pulmonary flow. The changes in flow relationships can be well demonstrated by the analogy of an electrical model with two parallel resistances, one of them variable.

McCord, van Elk and Blount (1058) make the cogent observation that the tetralogy of Fallot encompasses a broad spectrum of physiologic changes and a correspondingly broad clinical spectrum. In the opinion of these workers pulmonary stenosis and ventricular septal defect are the essential lesions and dextroposition of the aorta of secondary importance. They present evidence that there is not always equalization of pressure in the two ventricles. Because of the variability, T/F may masquerade as VSD with left to right shunt or as isolated pulmonary stenosis.

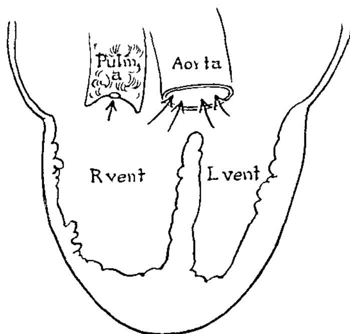


Fig. 383. The murmur of T/F is usually generated at the pulmonary stenosis.

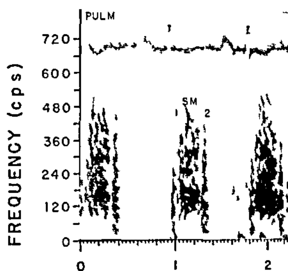


Fig. 384. Aortic area in J. S. (16075r) 43 year old female with clinically typical *tetralogy of Fallot* established by cardiac catheterization. Rather extreme hypertrophic pulmonary osteoarthropathy is present. S_2 unitary. Systolic murmur stopping slightly before S_2 as in aortic stenosis. Contrast characteristic pattern with that of pure pulmonary stenosis (e.g. Fig. 370).

Further light is shed on the interrelationship of T/F of the cyanotic and non cyanotic types (1318) with simple VSD by the findings of Gasul and colleagues (528). When infants who were found on first catheterization to have changes of simple VSD are recatheterized, evidence of a gradient from right ventricle to pulmonary artery and even reversal of the direction of the shunt may be found. This probably explains the fact that striking improvement in cases of VSD often occurs after the first year of life. Possibly the mechanism of the "acquired" pulmonary stenosis is hypertrophy of the crista supraventricularis.

CARDIOVASCULAR SOUND. The systolic murmur is probably produced (1495) mainly or exclusively at the stenotic pulmonary outflow area (Fig. 383). Because of the overriding of the aorta there is probably little obstruction to outflow through the aorta; therefore a murmur is not likely to arise at the VSD. Supporting the view that the pulmonary stenosis is the generator of the murmur are the facts that (1) in pulmonary atresia there is little murmur, (2) with thrombosis of the outflow tract, which occasionally develops, a pre-existing murmur disappears, and (3) a decrease in the

TABLE 15

The differentiation of tetralogy of Fallot from trilogy of Fallot (pure pulmonary stenosis with patent interatrial septum)

Points of Differentiation	Tetralogy of Fallot	Pulmonary Stenosis with Intact Ventricular Septum and Interatrial Communication
Sounds		
Systolic murmur	Stops before aortic component of S_2	Extends to or past aortic component
Pulmonary component of S_2	Usually absent	Usually present in delayed and diminutive form (pulmonary reversal snap)
Systolic ejection sound (early systolic click)	Frequently present; originates in aorta relatively late in systole; called wide splitting of first sound	Present in mildest cases only; originates in pulmonary artery relatively early in systole; called close splitting of first sound
Aortic component of S_2	Often accentuated in pulmonary area	Usually unchanged
EKG	Extreme right ventricular hypertrophy and strain rare	Extreme right ventricular strain may occur
Venous pulse	Giant a waves never present; Dominant a waves unusual	Dominant or giant a waves the rule
Radiography		
Heart size	Usually not increased	May be increased
Pulmonary artery	Small	Full from poststenotic dilatation
Aorta	Wide; right aortic arch in 25%	Relatively normal; right arch very rare
Heart shape	Sabot; elevated apex; concavity of mid left heart border	
Palpation	S_2 often palpable in aortic area; often systolic thrust in pulmonary area from anteroposed aorta; thrill often absent	Sustained lift over RV outflow tract; thrill usually present
Cyanosis	May develop late but usually before 18 mos	Usually develops late
Squatting	More common (about 50%)	Occurs less often (about 15%)
Angina pectoris	Very rare	Relatively common

Adapted from data of Vogelpoel et al. (1496) and Campbell (2411).

in the extreme case in which it is a systemic ventricle, must begin to relax before the aortic valve can close. The murmur generated at the stenotic pulmonary valve will cease before the aortic valve closes. One might think of these relationships in terms of pressure and murmur threshold. The threshold of pressure for generation of murmur is likely to be higher than the threshold for opening (and closing) of the aortic valve. With the rise of intraventricular pressure the aortic valve will open before the onset of the murmur; with fall in intraventricular pressure the murmur will cease before the aortic valve closure.

The fact just stated is useful (1495) in differentiating T F from PS with right-to-left shunt at the atrial level (compare Figures 384 and 385). Other points helpful in this differentiation are presented in Table 15. Two reports (866, 1496) which appeared almost simultaneously are in agreement on most of the descriptive aspects of T F as opposed to PS with interatrial communication—a possibly confusing problem in clinical differential diagnosis (430). However they disagree on how characteristic of each condition is the duration of the systolic murmur. Thus scrutiny of the cases of Vogelpoel and co-workers (1496) appears to indicate that the systolic mur

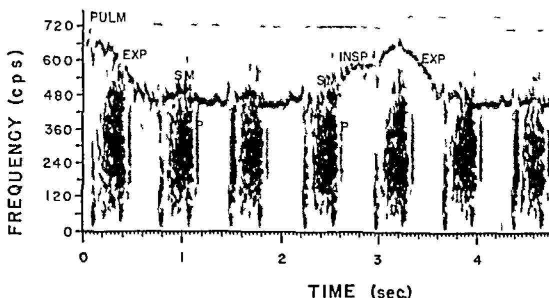


Fig 385 C Valvular pulmonary stenosis

Pulmonary area in M K P (773401) 14 year old girl confirmed at operation Right ventricular pressure was 137 mm systolic The characteristic late peaking of the systolic murmur and the diminutive pulmonary reversal sound are well demonstrated

resistance seems to *favor pulmonary flow* Clinically, the patients do better than do patients with only T/F of similar anatomical type (4) Finally there is a rather satisfactory correlation between the volume of pulmonary valve *flow* and the *in*tensity and duration of the systolic murmur of pulmonary stenosis In pulmonary stenosis with intact septum pulmonary valve flow has in the experience of most laboratories been approximately twice that in cases of T/F

Sometimes, because of the fact that the aorta is carrying almost all, or in fact all of the cardiac outflow a murmur of high flow is generated in the aorta An early systolic click (ejection sound) arising in the aorta may occur for a similar reason (989) The ejection sound usually occurs about 0 07 sec after the first sound It is most likely to occur after Blalock Taussig or Potts procedure, because flow through the aorta is augmented The ascending aorta is likely to be closer to the sternum than normally this favors transmission of sounds generated in the aorta Furthermore, the enlargement of the ascending aorta emphasized by Pattinson and Emanuel (1185) may be a factor in the systolic click That the sound does not arise in the pulmonary artery is attested by its presence in cases of T/F with *pulmonary atresia Furthermore the usual lack* of dilatation of the pulmonary artery makes this structure an unlikely source of the sound Still further Leatham and Weitzman (866) observed a case *in which the murmur (arising presumably* at the pulmonary stenosis) began before the sound (arising presumably in the aorta)

Although the murmur of Fallot s syndrome is generated at the pulmonary stenosis it usually differs from that of PPS It is variable in its intensity from case to case It may stop with a brief silent gap before the aortic closure sound (e g Fig 384), rather than running up to the aortic closure sound as in the case of the murmur of PPS The peak of the Christmas tree murmur occurs earlier in systole The basis for the difference in the heart sound pattern of T F from that of uncomplicated PPS is not the fact that most PPS is valvular whereas infundibular stenosis predominates in cases of T/F Rather the VSD of T/F is fundamentally responsible for the difference In aortic stenosis the systolic murmur stops slightly before the aortic closure sound because obviously contraction of the ventricle must relax a bit before the aortic valve can close and the murmur will cease with relaxation of the ventricle In T/F the right ventricle especially

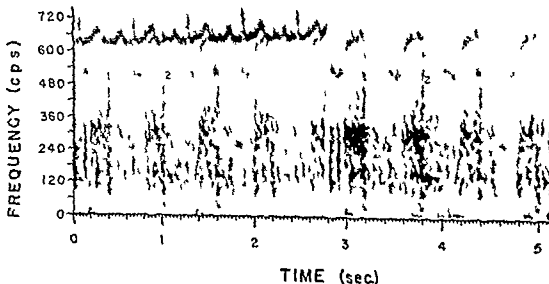


Fig. 357. Musical murmur from bronchial collaterals in T/F.

Right infraclavicular area in A. N. (a. 56), 6 year old female with typical tetralogy of Fallot except for the additional finding shown here. There is a right aortic arch. Over the right upper chest there is a continuous murmur with musical element consisting of a single harmonic which describes an arterial pulse pressure curve. Probably from dilated bronchial arteries which are serving as collateral to the lung. In the display on the right the EKG was attenuated for better definition of the harmonic of the musical murmur.

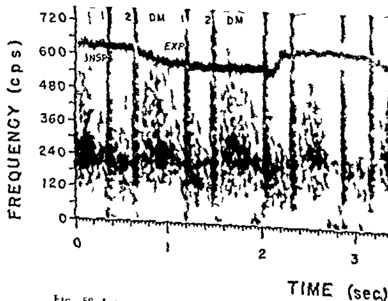


Fig. 58. Late complication of Blalock Taussig in T/F.

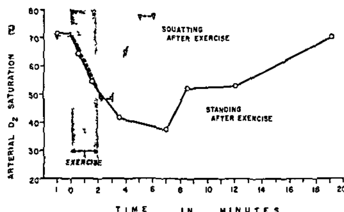
Pulmonary area in H. (3479) 18 year old male with tetralogy of Fallot who was the 18th patient to have the Blalock Taussig operation (right pulmonary subclavian anastomosis) 11 year before the recording. Before operation there was only a systolic murmur at LSB. Postoperatively a continuous murmur was maximal in the ... and hematocrit fell from 56 to 4... percent. Ten years after operation a diastolic murmur was heard ... dilatation of pulmonary arteries was described. The continuous murmur had disappeared and the patient was bothered by cough with occasional small hemoptysis. The decrescendo diastolic murmur is probably that of pulmonary regurgitation. The gap between ... closure and the onset of the murmur probably ... that which usually separates aortic and pulmonary ... sound in pulmonary ...

mur followed the pattern outlined above in 13 of 14 cases of PPS with interatrial communication and 39 of 48 cases of T/F. Leatham and Weitzman (866), on the other hand, found that the systolic murmur stopped before the aortic closure sound in only 7 of 26 cases of T/F.

Vogelpoel (1495) thought the presystolic murmur which occurs in some cases of PPS and the loud atrial sound which occurs in others might be helpful in differentiating T/F from PPS with ASD. However, Leatham and Weitzman (866) found an auricular systolic murmur in two patients with T/F and large *a* waves in the neck.

In T/F the second heart sound tends to be unitary—exclusively aortic closure sound. So called "P2" may be normally loud or possibly even increased. The aortic closure sound is unusually well heard in the pulmonary area because of the anatomic reorientation of the aorta. Furthermore, unlike PPS, in which a diminutive, delayed pulmonary closure (or reversal) sound is demonstrable, at least by graphic means, in about 85 per cent of patients, in Fallot's syndrome the incidence is about 15 per cent. Vogelpoel and Schrire (1495) found it in 3 of 18 patients. When present it should be suspected that pulmonary flow is greater than the average. Clinically, the patients are less incapacitated. The sound may appear after Blalock-Taussig operation or direct repair of the outflow tract. Leatham and Weitzman (866) describe an instructive patient in whom the pulmonary closure sound first appeared after Blalock-Taussig operation, located 0.11 sec. after the aortic component. After subsequent valvotomy the interval was reduced to 0.06 sec. and an early diastolic murmur appeared following the pulmonary sound and displaying a crescendo-decrescendo pattern.

Vascular bruits, sometimes continuous, presumably arising in the bronchial artery collaterals



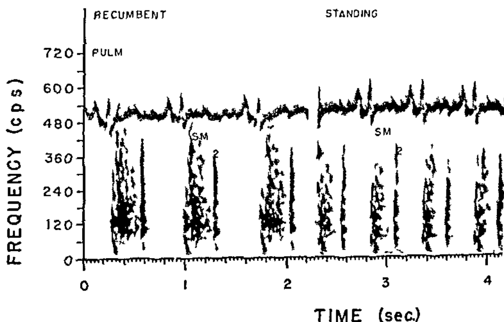


Fig. 386. (*Above*) In T/F the effect of exercise with and without squatting on the oxygen saturation of arterial blood. (From Lurie (996).) (*Below*) Decrease in murmur of T/F when the patient (D. K. 370059) is standing is compared with the recumbent position. Recordings made with identical amplification. The effect of exercise followed by quiet standing on the murmur of T/F in the same patient.

stenosis and tricuspid atresia need no particular elaboration. Congenital tricuspid stenosis can occur as an isolated valve lesion, unlike the rheumatic form of the disease which almost always is accompanied by disease of the mitral and/or aortic valves. Essential to survival in tricuspid atresia is the existence of an interatrial communication (so that venous blood can have access to the left side) and usually also a VSD for transmission of blood to the lungs.

PHYSIOLOGIC CONSIDERATIONS. In the Ebstein's malformation there is the anomalous functional situation such that part of the wall of the pretricuspid chamber contracts at the same time as does the ventricle. Because of the deficiency in the ordinary right ventricle there is impairment in the propulsion of blood into the pulmonary circulation. Furthermore, there is circumstantial evidence from cardiac catheterization as well as from study of the murmur that contraction of the ventricular component of the pretricuspid chamber contributes to right sided ejection. Specifically, pressure curves from the pulmonary artery show a secondary rise in the latter part of the period of ventricular systole, synchronous with a similar rise in pressure in the pretricuspid chamber (108, 119).

Cyanosis may be present if there is an interatrial communication. The ECG shows large notched P waves and there is usually pulsation of the neck veins and liver. A left parasternal heave is conspicuous by its absence. In the diagnosis simultaneous intracardiac ECG and pressure tracings by means of a double cardiac catheter may be helpful for a reason which will probably be obvious, although usually the other features listed here are adequate for a satisfactory clinical diagnosis and cardiac catheterization may be hazardous (1, 1690). Often there is rather remarkably good exercise tolerance in relation to the size of the heart, the impressiveness of the gallops and the degree of cyanosis. Survival may be long, to the age of 79 years in one case (7). Recognized with increasing frequency in adults, Ebstein's anomaly is a variety of congenital malformation which is frequently mistaken for rheu-

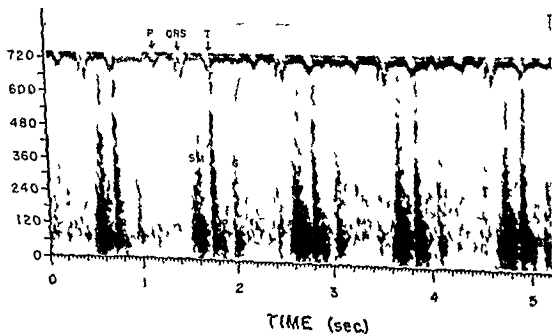


Fig. 330. Ebstein's anomaly.

Recorded in left midprecordium in 14 year old male G. R. (A503,41). Right bundle branch block and prolonged P-R interval are present. The first sound (1) is faint. There is a mid systolic murmur (SM) with the configuration of a systolic ejection murmur. The second component of the split second sound, probably pulmonary closure (P), is diminutive relative to the first (A). A protodiastolic gallop (G) is present and probably initiates a short murmur.

to the lungs, may be audible They are often so striking as to suggest the diagnosis of patent ductus arteriosus Occasionally these bruits are musical (see Fig 387)

The creation of an artificial ductus arteriosus by the operation of Blalock and Taussig or that of Potts results in a continuous murmur with the graphic configuration characteristic of intrathoracic arteriovenous fistula Abbreviation or disappearance of this murmur is a clue to the fact that obliteration of the anastomosis is occurring More and more patients in whom Blalock Taussig or Potts operations were performed ten or more years ago are having return of cyanosis and disability In many of these criteria other than the murmurs must be used in evaluation of the original anastomosis because the murmur of bronchial collaterals may be indistinguishable from that of the anastomosis

A pulmonary early diastolic murmur may develop after operation Such is more common after direct operation but interestingly may occur after shunt operations, particularly if an excessively large shunt is created with subsequent dilatation of the pulmonary artery (Fig 388)

TRICUSPID LESIONS

(including Ebstein's malformation, tricuspid stenosis (900), tricuspid atresia)

ANATOMIC CONSIDERATIONS (1416) Ebstein's malformation (398, 410, 792, 1091) consists of a downward displacement of the tricuspid valve such that part of the right ventricle becomes incorporated into the right atrium, or, at least, into the pre tricuspid chamber The tricuspid valve is usually deformed in the way of fenestration double orifice (800), etc so that one would from the necropsy appearance, anticipate both regurgitation and some obstruction to forward flow In about half of all cases there is an associated interatrial communication The pre tricuspid chamber becomes huge The pulmonary artery, on the other hand is usually small

Description of the anatomic state in tricuspid

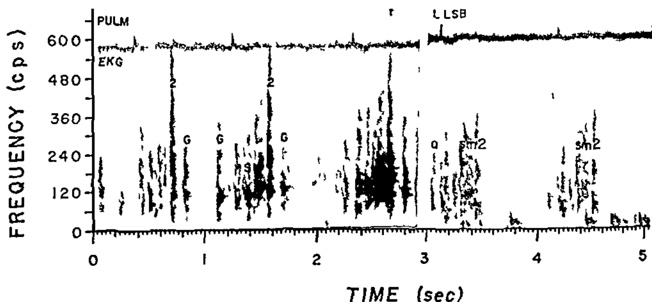


FIG 389 Ebstein's malformation

R J T (A91519) 11 year old white male has from birth been cyanotic and dyspneic on exertion with quitting and fainting spells Angiocardiogram at age of 7 showed very large and slowly emptying right atrium with large ASD EKG reveals large P wave with PR of 0 28 sec and right bundle branch block The PR interval became normal after the longer diastolic periods of sinus arrhythmia

SPCGS There are both presystolic and protodiastolic gallops and a mid or late systolic murmur which has the appearance of an ejection systolic murmur and which may be caused by forward movement of blood through the tricuspid valve The atrial gallop is at times a short murmur and because of a long PR interval is usually in mid diastole In the last cycle (pulmonary area) the PR is shorter after a longer diastolic pause and the atrial sound is closer to the first heart sound In some areas the protodiastolic sound is snapping as indicated by its pure frequency content at about 200 c p s This is probably caused by snapping of the tricuspid apparatus in early diastolic inflow

CARDIOVASCULAR SOUND. Among 82 patients with the Ebstein malformation reviewed by Kilby *et al* (792) there were only 10 without murmurs, systolic and/or diastolic. The second important feature (and among congenital malformations a feature rather characteristic of the Ebstein malformation) is the presence of striking diastolic gallop sound. These were present in 30 of 82 patients (792). Often a quadruple rhythm results from the presence of both third sound and fourth sound gallop. Although more will not be said about the gallop sounds it should be emphasized that they are perhaps the most important auscultatory feature of the Ebstein anomaly.

A systolic murmur is almost always present. Often it has been attributed to tricuspid regurgitation. The anatomic change in the valve and the graphic demonstration of a holosystolic murmur (Fig. 392) are in accord. This murmur is likely to be audible at the usual position of the cardiac apex because the tricuspid valve is situated further to the left than is ordinarily the case.

At other times the systolic murmur is circumscribed and late and has the configuration of an ejection stenosis murmur. This murmur may result from contraction of the ventricular myocardium in the wall of the pre-tricuspid chamber with forcing of blood through the malformed tricuspid valve. See Figures 390 and 391 for an SLCC of this phenomenon and see Figure 499-4 of Kjellberg, *et al* (800) and Figures 1 and 3 of Kilby *et al* (792) for oscillographic displays of the seemingly identical phenomenon.

In still other cases the sound in systole is more like a sound (in the usual sense) than a murmur. This sound may likewise be in the last analysis caused by contraction of the ventricular component of the wall of the pre-tricuspid chamber. In essence it may be an atrial heart sound occurring in ventricular systole because of the particular anatomic and physiologic [illegible] Ebstein's malformation.

Usually the diastolic murmur(s) of the Ebstein anomaly similarly have origin at the malformed tricuspid valve. The murmur may be middiastolic or more often presystolic (i.e., atrial) and is usually best heard at the left sternal border. It is appropriately referred to as an atrial murmur since the PR interval is often long and the murmur not strictly presystolic. It was present in five of eleven cases at the Mayo Clinic (792); five authors have presented phonocardiograms showing this phenomenon in a total of about seven patients (63, 669, 792, 1091).

At times (probably more often than is indicated by the reports in which it is specifically mentioned) the systolic and diastolic murmurs assume a to and fro character (Fig. 392). Together with a superficial scratchy quality of the murmurs, a cardiac contour suggesting pericardial effusion and quiet heart borders on fluoroscopy, this feature may lead to a mistaken diagnosis of pericardial disease (429). Wood (1590, p. 5[illegible]) speaks of a very characteristic superficial diastolic scratch which sounds more like a diastolic pericardial friction over the distended right atrium than a true intracardiac murmur.

CONGENITAL MITRAL STENOSIS

ANATOMIC CONSIDERATIONS. Endocardial fibroelastosis is often associated with congenital mitral stenosis; the mitral stenosis is in such cases apparently one manifestation of the pathologic process. Congenital mitral stenosis may occur in association with other cardiovascular malformations such as coarctation of the aorta. With atrial septal defect in Lutembacher's syndrome the mitral stenosis is usually rheumatic but may be congenital. There are a few reports of a supravalvular or prevalvular variety of congenital mitral stenosis (363, 408, 1306) (p. 133 of ref. 152). This condition is often referred to as cor triatriatum for obvious reasons. Most of the cases have had hemodynamic changes typical of mitral stenosis but none of the auscultatory changes (1352A).

Mitral stenosis as an isolated congenital malformation is rare. It occurs more often in combination with other malformations. Regurgitation is much more frequently the functional result of a congenital mitral malformation.

PHYSIOLOGIC CONSIDERATIONS. In terms of the pathologic physiology of the valvular lesion there is no significant difference between mitral stenosis of rheumatic and congenital origin. There may, of course, be associated myocardial or other valvular lesions in the case of the rheumatic disease and associated congenital malformations in the case of the congenital disorder.

matic heart disease especially when it is not accompanied by cyanosis Reifenstein *et al* (1253) described a 20 year old Marine with Ebstein's malformation Possibly the good prognosis should not be overemphasized, however The long survivals cited above are an expression of variability of the clinical picture in this malformation sudden death occurs in this group

In congenital tricuspid stenosis, the pathologic physiology does not differ significantly from that in the acquired form of the disease except in connection with the fact that when *congenital*, tricuspid stenosis may present a less clouded picture because of the absence of lesions in other valves

In tricuspid atresia, cyanosis always occurs because of the interatrial right to left shunt essential to life

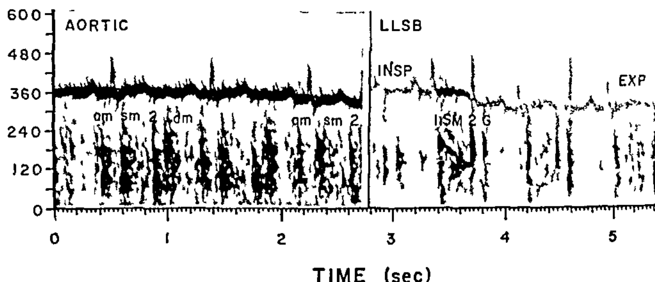


Fig 391 Ebstein's anomaly

K S (763208) born in 1940 had moderate cyanosis from birth until the age of 5 or 6 years She was however considered entirely normal until mid 1956 when she began having episodic precordial pain—her only complaint *The diagnosis of Ebstein's malformation was suggested by the auscultatory findings* long PR interval low voltage of QRS's on right side of precordium incomplete right bundle branch block prominent right atrium with small pulsations

The scratchy quality of the murmur is rather characteristic of Ebstein's disease The systolic murmur is holosystolic *suggesting tricuspid regurgitation*

On cardiac catheterization the right atrium was demonstrated to be large and the tricuspid valve to be displaced well to the left of the midline There was a double hump in the pulmonary artery pressure tracing Femoral unsaturation (as well as the polycythemia) suggested atrial septal defect

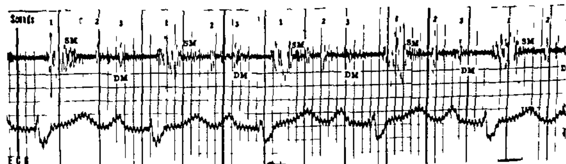


Fig 392 Ebstein's anomaly

Prominent first sound (1) well split second sound (2) prominent third sound (3) decrescendo medium frequency *moderate intensity* systolic murmur (SM) mid diastolic murmur (DM) following the third sound and the P wave of the electrocardiogram (Courtesy of Ongley (1159) and *Circulation*)

Fig 394 Congenital mitral stenosis in R. F (B36036) age 17 months. In the pulmonary area there is an early systolic click followed by a decrescendo murmur of ejection type. The presystolic murmur can be identified here as is often the case in rheumatic mitral stenosis in young persons or any patient with a small chest. There appears to be an opening snap. At the apex S_1 is snapping. The passive diastolic murmur is introduced by a rather clicking sound which is later than the sound interpreted as opening snap in the pulmonary area. This is probably an unusually clicking variety of the sound which often follows the opening snap and imitates the diastolic rumble in mitral stenosis

of the aorta a diastolic rumble at the apex is often heard. For example Wood (1950 p 355) states that he heard such a murmur in one third of cases of coarctation. Although actual congenital malformation of the valve may be present in some in the majority of these dilatation of the ventricle and relative mitral stenosis seem likely. Endocardial fibroelastosis a rather frequent association with coarctation may contribute to the production of a diastolic murmur

In brief the auscultatory diagnosis of congenital mitral stenosis is difficult because *on the one hand* the characteristic diastolic murmur may be absent and on the other closely simulating signs are produced by so called relative mitral stenosis. The presence of an opening snap strengthens the diagnosis but is not entirely pathognomonic

Other congenital malformations of the mitral valve include cleft in association with ASD of the ostium primum type (p 350) Ebstein type malformation (78) fibroelastotic change in the valve mechanism without significant obstruction. The last may occur alone or in association with other malformations particularly congenital aortic stenosis or coarctation (q v). Cleft of the anterior leaflet of the mitral valve may occur alone (408) although it is much more often associated with endocardial cushion defect. This and anomalous insertion of the chordae tendineae represent forms of congenital mitral regurgitation

CONGENITAL AORTIC STENOSIS

ANATOMIC CONSIDERATIONS Subvalvular valvular and supravalvular varieties of congenital aortic stenosis are described. Therefore essentially the same three varieties exist as in the case of congenital pulmonary stenosis. We have observed a familial incidence (in grandmother and grandson) of the supravalvular variety (1140). The difficulties of differentiating congenital and rheumatic (or other acquired) bases for aortic stenosis are not only clinical but also—to a considerable extent—anatomical as well. The subaortic and supravalvular types of the disease can of course be identified as congenital at least at necropsy. There is usually about one subaortic case for each four valvular cases of congenital aortic stenosis. In the valvular type the commissure between the *left* and non coronary cusps is usually least affected. Early recognition of a murmur consistent with aortic stenosis is the usual clinical criterion for congenital etiology e.g. a murmur heard before the age of four years is taken as the criterion by some. Occasionally both congenital and acquired factors are importantly involved in the causation of aortic stenosis. For example bicuspid aortic valve when it occurs

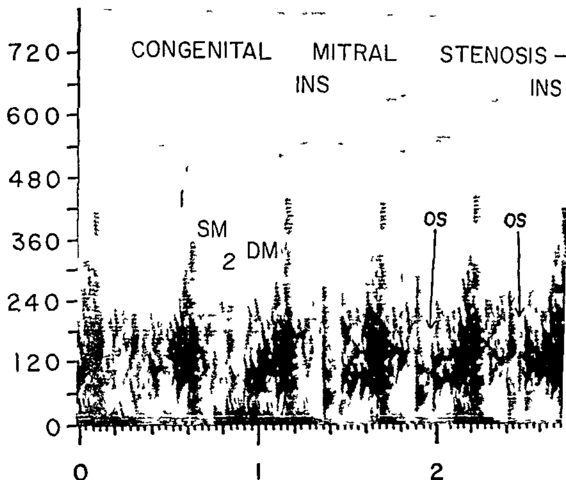


Fig. 393 Congenital mitral stenosis

Recorded at apex in C. C. (B15690) one year old child with clinical, operative and autopsy findings of mitral stenosis. Apex. Note the ringing M_1, probable opening snap (OS) and typical diastolic rumble with both passive and active (presystolic) components. Compare with rheumatic mitral stenosis (e.g. Fig. 274).

CARDIOVASCULAR SOUND. In the two well confirmed cases of isolated mitral stenosis that I have studied (Figs. 393 and 394) there has been, judging by the presence of a systolic murmur at the apex and the findings at operation or autopsy, some degree of mitral regurgitation. In some patients the changes otherwise have been virtually identical to those in "pure" mitral stenosis of rheumatic origin. For example take the one year old child whose murmur is portrayed in Figure 393. There is a snapping first heart sound and a diastolic murmur which begins following a short gap after the second sound and has a crescendo into the first heart sound. There may even be a faint opening snap but it is difficult to be certain because of the rapid rate. In the second case (Fig. 394) the *opening snap* is more definite.

Difficulties of differential diagnosis are presented by various congenital malformations which because of altered hemodynamics and/or anatomy result in a rumbling diastolic murmur of relative mitral stenosis. Conditions of high flow across the mitral orifice—VSD, PDA—represent one group. The diastolic murmur of high mitral flow as opposed to that of structural stenosis is usually limited to mid diastole and has no presystolic accentuation. It often is initiated by an accentuated third heart sound. It has, therefore, the features of a Carey Coombs murmur (p. 199). The tricuspid high flow murmur in ASD may be loudest at the cardiac apex and may be distinguished with difficulty from that of mitral stenosis.

Duplication of the mitral valve, that is, double orifices with reduction in total orifice area, has been described (333, 1366, 1559) in association with mild coarctation and calcific aortic stenosis. An apical mid diastolic murmur was *produced* (1559).

In congenital aortic stenosis and in *coarctation*

Fig. 398. Congenital aortic valvular disease.

H. L. (63 50 11 I H 79) a 21 year old male has been seen at this hospital from the age of [illegible] year, making the congenital nature of his lesion quite definite. Furthermore, the mother has aortic stenosis with no history of rheumatic fever and the grandmother was found at autopsy to have aortic stenosis, almost certainly congenital (1100). A sister has a small PDA. In this patient VSD or Eisenmenger complex had long been thought most likely as often happens because of the localization of the murmur at the left sternal border. Left heart catheterization in 1959 revealed a pressure of 154/36 mm Hg in the left ventricle and 83/73 (?) in the aorta. The SECG from both areas shows changes typical of AS and AR. The diastolic murmur has an unusually narrow frequency span giving it a somewhat musical quality.

Fig. 399 and 398. Congenital aortic stenosis with apical diastolic rumble.

Aortic area and apex in J. R. W. (B-31 [illegible]) [illegible] year old male. There is a typical murmur of aortic stenosis in both areas. At the apex there is a third heart sound followed by a short rumble which was quite definite by stethoscope. The rumble had not been present a year before.

ECG. As noted before, the quality of A_2 is a poor index of the severity of aortic obstruction; it is also a poor index of the type of malformation which is present.

It was formerly thought that significant aortic regurgitation occurs uncommonly with congenital aortic stenosis. It is now clear that this is not true (e.g., Fig. 396).

Wood (1500) states that one sixth of his patients (totalling 30) had an apical diastolic rumble suggesting mitral stenosis, either relative or structural. The possibility of fibroelastosis

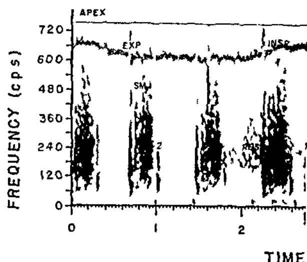


FIG 395 Congenital aortic stenosis

B C W (741719) 11 year old male had had progressive evidences of left heart strain and a bout of SBE nine months previously Right heart catheterization revealed only normal findings

is an isolated malformation or in association with coarctation of the aorta is prone to undergo atherosclerotic change (269) probably because of the disturbed flow pattern in the vicinity of the valve Bacterial endocarditis to which bicuspid valves are prone is likely to lead to distortion of the valve calcification may occur in a valve so affected Furthermore without bacterial endocarditis indubitably congenital aortic stenosis may show calcification of the valve and clinically show an evolution to suggest that a progressive atherosclerotic process is occurring

Clinically congenital aortic stenosis behaves like a progressive lesion With the passage of years the murmur becomes progressively louder and the manifestations of left ventricular strain become more pronounced This phenomenon was well demonstrated in the following patient W P (461996) white male had been observed at frequent intervals from the age of 17 months when he was hospitalized for a prolonged diarrheal illness During this hospitalization many examinations made no mention of a murmur only two recorded a soft apical systolic murmur During the next decade the systolic murmur which first became impressive when the patient was 4 years old displayed a "march" from the apex to the left sternal border and finally to the aortic area (as point of maximum audibility) and rheumatic heart disease with mitral regurgitation was entertained as the leading possibility The electrocardiographic, radiologic, and clinical evidences of left ventricular hypertrophy and "strain" were progressive At open heart surgery when the patient was 18 years old, a diaphragmatic subaortic stenosis was discovered

In a case such as this it seems likely that the obstructing diaphragm does not grow (at least its orifice does not enlarge) commensurate with general and cardiac growth and with steadily increasing cardiac output The murmur heard in earlier years at the apex, left midprecordium and left sternal border may be generated at the immediate area of obstruction As the gradient across the aortic valve area became greater, the jet in the aorta became more pronounced and the resulting murmur in the aortic area appeared

Experiences such as this make one hesitant to render an unequivocal judgment of benignity in the case of relatively faint murmurs in young children

Post stenotic dilatation of the ascending aorta is clinically demonstrable in the great majority of instances if sought by proper radiologic methods Occasionally this phenomenon attains mammoth proportions

PHYSIOLOGIC CONSIDERATIONS See pp 263 and 264

CARDIOVASCULAR SOUND I will speak mainly of those features which tend to be different in congenital aortic stenosis than in the familiar rheumatic or calcific aortic stenosis of adult life Obviously none of these features is an absolute or pathognomonic differential point otherwise there would not be the usual difficulties in differentiating congenital from acquired aortic stenosis

Often especially in young subjects and in subaortic stenosis the murmur is loudest at the left sternal border and leads to the mistaken diagnosis of VSD

Brofman and Feil (184) thought that subvalvular stenosis would be distinguished from valvular stenosis by the presence of a good A in the former variety Kiloh's experience (793) was not in accord with this view, however and Mannheimer (1033) described an autopsy confirmed case of subvalvular stenosis in which no aortic second sound was detected by stethoscopy or

mechanisms of the sound come to mind (1) an opening sound produced at the aortic valve (2) a snapping of the wall of the aorta dilated through the phenomenon of post stenotic dilatation The sound is probably comparable to that which occurs in milder cases of pulmonary stenosis (p 370)

IDIOPATHIC DILATATION OF THE PULMONARY ARTERY

ANATOMIC AND ETIOLOGIC CONSIDERATION Care must be taken to exclude dilatation of the pulmonary artery secondary to ASD or mild PS. Occasionally cardiac catheterization forces one to conclude that pulmonary artery dilatation is an isolated malformation A certain number probably small of these cases may have dilatation of the pulmonary artery as one manifestation and perhaps the main one of the Marfan syndrome (1071)

Greene *et al* (589) pointed out that it is possible to demonstrate a pressure drop across the pulmonary valve with only idiopathic dilatation of the pulmonary artery

CARDIOVASCULAR SOUND An early systolic click and a systolic murmur are to be expected The loss of pressure energy as indicated by a pressure drop across the pulmonary valve accords well with the fact that a murmur is present Occasionally a diastolic murmur is heard and attributed to relative pulmonary insufficiency (561) The second pulmonary sound tends to be loud probably caused mainly by improved conduction of the sound to the front of the chest possibly in part to closure of the valve from a more wide open position

PRIMARY PULMONARY HYPERTENSION

To what extent this condition can be considered congenital is not clear See pages 428 to 432 for a discussion of this entity and of pulmonary hypertension in general

CARDIOVASCULAR SOUND The changes of note are (1) accentuated pulmonary second sound (2) early systolic click (3) presystolic gallop (4) pulmonary diastolic murmur

QUADRICUSPID SEMILUNAR VALVE

As discussed on p 107 quadricuspid state is more frequently encountered in the pulmonary valve than the aortic Furthermore for reasons elucidated by Leonardo da Vinci about 1500 and in 1873 by Longworth (see p 36) a four cusp valve is less strong in the closed position than a three-cusp valve Pulmonary regurgitation may occasionally develop (798) Probably this is especially likely to occur if pulmonary hypertension is present for some reason

The early diastolic murmur of pulmonary regurgitation in this condition when unaccompanied by pulmonary hypertension may be unusually low pitched and blubbering This statement is based on the findings in patients in whom I suspect without anatomic confirmation that the diagnosis is quadricuspid valve Wells and co authors (1529) have presented a loud and unusually low pitched diastolic murmur in a patient with pulmonary regurgitation The low pressure in the pulmonary artery is consistent with a blubbering quality rather than the higher pitched more whirring quality of the Graham Steell murmur of pulmonary hypertension and the murmur of aortic regurgitation Furthermore the murmur may have a crescendo-decrescendo—rather than purely decrescendo—pattern

BICUSPID SEMILUNAR VALVE

ANATOMIC CONSIDERATIONS As discussed on p 107 bicuspid condition most often affects the aortic valve Although it may occur as an isolated anomaly it occurs frequently in association with coarctation of the aorta Abbott (1) found it in 23.5 per cent of 200 cases of coarctation *and* Reifenstein Levine and Gross (1254) found it in 42.3 per cent of 104 cases It is *frequently* difficult to distinguish a congenitally bicuspid valve from a valve rendered bicuspid through the rheumatic adhesion of two of its cusps (612) Particularly is this true when the atherosclerotic change to which both types of valve are prone has taken place When there is *a bicuspid* pulmonary valve it is usually in association with a major malformation such as tetralogy of Fallot However Ford and colleagues (460) described a case of isolated bicuspid pulmonary valve *Pulmonary* regurgitation was present

CARDIOVASCULAR SOUND A bicuspid valve does not open completely Therefore a systolic murmur

See references 808 809 810

accounting for the aortic lesion and also affecting the mitral valve to some extent has been mentioned (pp 385 and 387) Dilatation of the ventricle as at least a contributing mechanism is suggested by the experience illustrated in Figure 397 At the age of nine this patient had no apical rumble, but did have one year later In the patient illustrated in Figure 400 a mid diastolic rumble appeared at the apex in the interval of six years between two examinations

In 15 patients, Reinhold, Rudhe and Bonham Carter (1258) found a striking protosystolic click which was well heard at the apex where it simulated a split first heart sound At least two

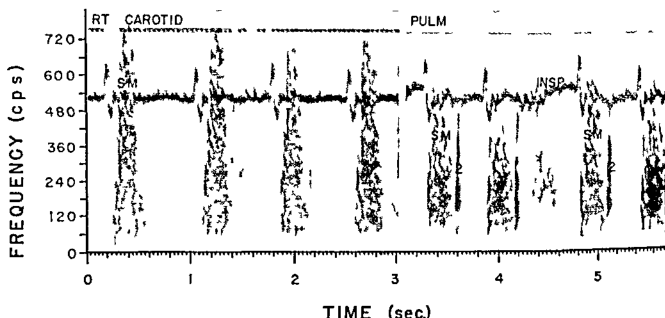


Fig 399 Congenital aortic stenosis with apical diastolic rumble

T D (733061) 3 year old male had a systolic pressure of 180 mm Hg in the left ventricle by left heart catheterization In addition to an early diastolic murmur suggesting mitral stenosis there was a presystolic gallop at the apex (not shown here)

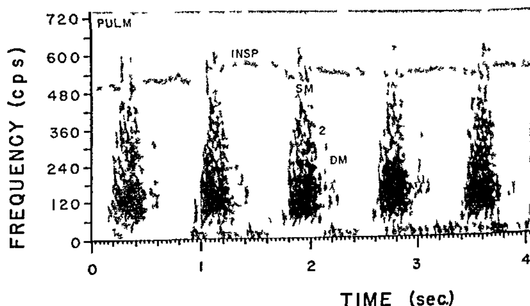


Fig 400 Congenital aortic stenosis and regurgitation are present in this 17 year old boy (S D I A90087) who has had a murmur from the first year of life Since being seen 6 years previously a mid diastolic murmur had appeared at the apex (not shown here)



Fig 401 Angiocardiographic demonstration of the location of coarctation of the aorta in four cases. ( ) dilatation of the ascending aorta is demonstrated in D. (Courtesy of Cooley and Sloan (791).)

area of the precordium, e.g., at LLSB. In such instances, origin at the coarctation can be determined by the late onset of the murmur, usually 0.12 sec or more after the first sound (see Fig 403).

The murmur of coarctation may extend into the very first portion of the diastolic period of the ventricle merely because of the lag in transmission of the pulse wave from the heart to the coarcted area. However, a continuous murmur or a murmur with long diastolic component probably has its origin in the collaterals or in an associated patent ductus arteriosus, not in the coarcted area itself. Another theoretical explanation for a murmur which extends into diastole is the principle of Myers *et al* (1176). If mean pressure is suffi

is to be expected As a result of secondary change in the valve, of atherosclerotic and calcifying character, a more pronounced *systolic murmur* and in addition a diastolic murmur may develop A bicuspid valve is probably not as strong in the closed position as a tricuspid, however, regurgitation is not likely to develop in the absence of hypertension and/or secondary atheromatous or bacterial change in the valve

The systolic murmur of uncomplicated bicuspid valve is not likely to be impressive Congenitally bicuspid arterial valve is nearly asymptomatic until complicated by atherosclerosis, bacterial endocarditis, or hypertension (1473)

COARCTATION OF THE AORTA

(Syn Coarctation of the aortic isthmus stenosis of the aortic isthmus)

ANATOMIC CONSIDERATIONS The most frequent site of obstruction is just distal to the ostium of the left subclavian artery in the first portion of the descending aorta (Fig 101) This site lies in apposition to the posterior chest wall— a point of importance in connection with the radiation of the murmur The mouth of the left subclavian artery may be narrowed or the right subclavian may arise independently of the innominate distal to the coarctation and pass to the right behind the esophagus Collaterals providing anastomotic flow between the proximal aorta and *the portion of the body beyond the coarctation* are striking and account for notching of the ribs and the visible pulsations over the neck and trunk Post stenotic dilatation occurs and is often apparent on ordinary chest x rays as part of the "figure of three" sign The coarctation may be for practical purposes complete Abbott in 1928 and Reifenstein Levine and Gross in 1947 found complete obliteration of the aorta in approximately one fourth of cases However Edwards (580, p 451) states that usually an opening can be demonstrated microscopically in all cases At the other extreme the narrowing may be very slight take for example, the mild coarctation that frequently accompanies other more striking features of the Marfan syndrome (1071) The caliber of the aorta at the coarctation is rarely more than 2 mm (I D) in surgical specimens A jet lesion, i e , a localized patch of atherosclerosis is sometimes seen distal to the coarctation *Progressive obliteration of the coarcted area* also occurs at times Dilatation of the ascending aorta proximal to the coarctation may occur on the basis of cystic medial necrosis, and dissecting aneurysm may develop sooner or later as a fatal complication

The high incidence of bicuspid aortic valve (269) with coarctation is previously mentioned Subaortic stenosis was found in 6 per cent of cases by Abbott (1) Fibroelastosis also occurs fairly frequently and may produce deformity of the mitral valve (see page 385) Mitral regurgitation and coarctation is a relatively frequent combination

Coarctations anatomically unusual in terms of length and site in the aorta are being encountered with increased frequency (728 737 771, 812)

PHYSIOLOGIC CONSIDERATIONS Often the flow through the coarcted area is by all evidence we have small, and, of course, occasionally there is no flow at all The characteristic feature is high pressure proximally and low pressure distally In the distal segment of the arterial tree mean pressure although lower than that in the proximal segment may be little below normal However, the pulse is greatly damped and the pulse pressure small

In experimental coarctation in dogs, Gupta and Wiggers (617) found that murmur developed *when the aorta was about 60 per cent constricted* was maximal at 73 per cent constriction, and was no longer audible beyond 78 per cent constriction

CARDIOVASCULAR SOUND (1530) The sonic phenomena include those referable (1) to the coarcted area itself (2) to the collateral circulation (3) to the associated aortic and other valvular lesions In addition an aortic early systolic click (ejection sound) is often heard

The murmur produced by the coarctation itself is likely *for obvious reasons to be loudest in the* upper portion of the left interscapular area It is usually well heard also in the left supraclavicular area and over the left upper chest anteriorly Graphically it has as one would predict the configuration of an ejection stenosis murmur

In some cases the primary murmur of coarctation is easily audible and recorded over a large

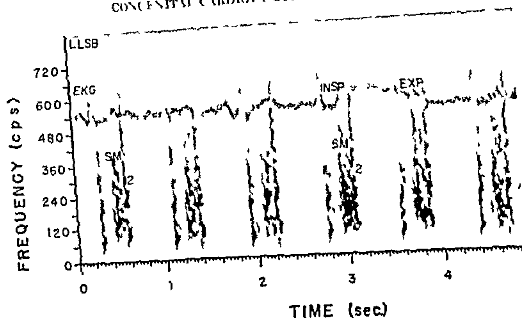


Fig 403 Coarctation

LLSB in V S (7760 6) 15 year old boy The late onset of the systolic murmur identifies it as the primary coarctation murmur There was a decrescendo early diastolic murmur which probably indicates aortic regurgitation Blood pressure was 205/70 mm Hg At the apex there was a sound with the temporal and spectral characteristics of an opening snap

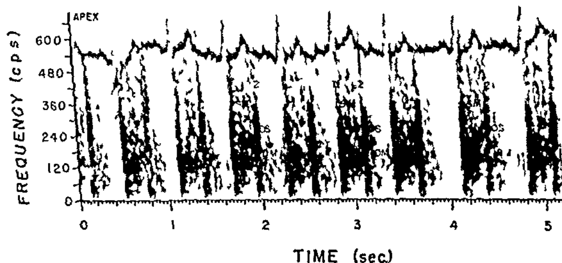


Fig 404 Mitral opening snap and apical diastolic murmur with *coarctation*

Apex in K B (40310) 6 year old boy Marked enlargement *of the left* atrium was demonstrated radiologically At operation for resection of the coarctation a systolic thrill was felt *over the large* tense left atrium at the site of the mural leaflet There was no diastolic thrill The left atrial pressure wave suggested predominant mitral regurgitation

efficiently may furthermore not be as strong in the closed position as a tricuspid valve An important factor in the aortic diastolic murmur in many cases is almost certainly dilatation of the base *of the* aorta with stretching of the ring seat *of the aortic* cusp Cystic medial necrosis may be the anatomic substrate of this dilatation in some instances (644) Aneurysm of the sinus of Valsalva

ciently lower distal to the coarctation than proximal, the murmur may extend into diastole and even a continuous murmur might be produced, merely because pressure proximal to the coarctation is higher during all parts of the cardiac cycle The view that extension of the murmur into diastole is due to backflow during that phase is untenable The primary murmur of coarctation is absent when the coarctation is complete and disappears with progressive obliteration Wood (1590) found no interscapular murmur in about 15 per cent of cases When the coarctation is at an unusual site the murmur may be heard in an anomalous position For example in cases of abdominal coarctation, the murmur was in the epigastric and upper lumbar regions (272, 737, 728, 771 812)

A continuous humming murmur with systolic accentuation arising in the collaterals may be present at various sites over the upper back At times this murmur may be only systolic with its onset possibly later in systole than the murmur generated at the coarcted area Occasionally it is possible to relate the murmur to a specific superficially located *anastomotic vessel the murmur* may be obliterated by pressure on the vessel (272) Sometimes the murmur in the back in the area of the coarctation is probably produced by blood entering the distal aorta in a collateral because the coarctation may be essentially complete (272) On the basis of a few patients who, albeit young and therefore subject to venous hums anyway, have had very striking murmurs of this *character in the neck (Fig 199), I have come to* view venous hums as a frequent feature of coarctation

An "aortic" systolic murmur occurs in the majority of cases of coarctation Causes, operative alone or in combination are (1) transmission of the primary coarctation murmur or murmurs of collaterals (2) bicuspid aortic valve, (3) calcific aortic stenosis (Fig 402), (4) subaortic stenosis, (5) dilatation of the aorta It is often exceedingly *difficult in a given case to* be certain which factor(s) is responsible (272) Campbell and Baylis (225) placed the incidence of aortic stenosis at 5 per cent

An aortic diastolic murmur occurs in a significant proportion of cases of coarctation In one series totalling 130 patients (225) such a murmur was present in almost one third It was present *in* 28 of 124 cases, excluding 6 with aortic stenosis (225) Wood (1590 p 338) found it in 10 per cent In 96 cases at the Mayo Clinic it was present *in 20 per cent That the hypertension is* at least indirectly responsible seems likely Secondary atherosclerotic change in a bicuspid valve may, with the hypertension account for the diastolic murmur in most cases the incidence and severity of aortic regurgitation increases with age (225) A simple bicuspid valve which does not open

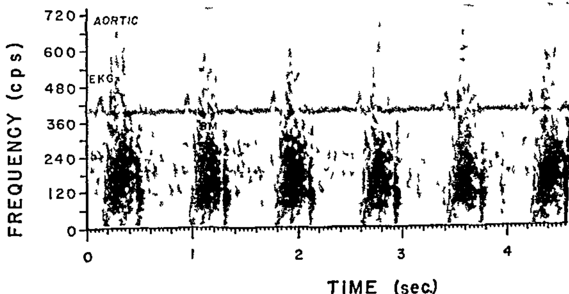


Fig 402 Aortic stenosis associated with coarctation Suprasternal notch in J W (762010) 53 year old male with coarctation The murmur is typical of aortic stenosis It occurs too early to be the murmur of the coarctation itself (Much background noise is present)

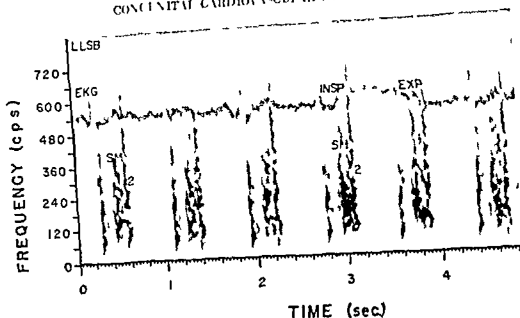


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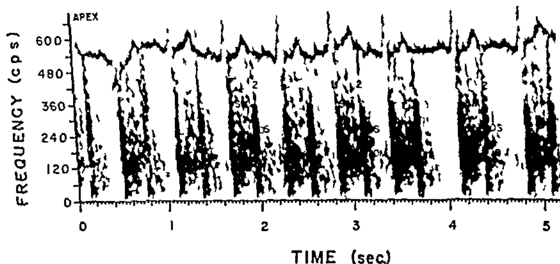


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has been demonstrated in association with coarctation (374), and a diastolic aortic murmur is likely to be present in such cases (560)

An important point is that when the aortic regurgitation associated with coarctation is of severe degree, as it sometimes is, the signs of coarctation may be submerged (for the unwary) The femoral pulses may feel quite adequate, if it is not appreciated that the water hammer pulse present in the arms is not felt there and that there is, in fact, a delay in the femoral pulse

Wood (1590, p 335) states that one third of his patients with coarctation had an apical diastolic murmur suggesting mitral stenosis In this enumeration he excluded five patients with associated VSD or PDA which alone may be accompanied by such a murmur and also excluded three patients with rheumatic heart disease and mitral stenosis Cleland and his colleagues (272) found the same apical mid diastolic murmur without other evidences of mitral stenosis in 15 of 40 operated cases They were impressed with its short duration less rumbling quality and inconstant nature Of the 15 patients two had persistent ductus arteriosus and six had an early decrescendo diastolic murmur at the left sternal border Size of the left ventricle could not be related to presence or absence of the murmur in any convincing manner In six of the patients the mitral murmur was not heard during post operative observations However, this notation is of doubtful significance because of the inconstancy of the murmur before operation We have noted (see Figs 404, 405, 406) the occurrence of what appears to be an opening snap in at least two patients with coarctation (P B, 775818, K B 740310) The coarctation was of classic type in these patients, the left atrium was enlarged in the first, however

The same explanations for the diastolic murmur (Figs 404 and 407) are applicable as in the case of the same murmur occurring with congenital aortic stenosis dilatation of the ventricle with relative stenosis or fibroelastotic change (1162) in the mitral valve with actual obstruction Actual mitral stenosis has been described with coarctation as an associated congenital malformation (741 1156)

Because of the hypertension and the frequent presence of dilatation of the ascending aorta, it is not surprising that an early systolic click is often heard in the aortic area

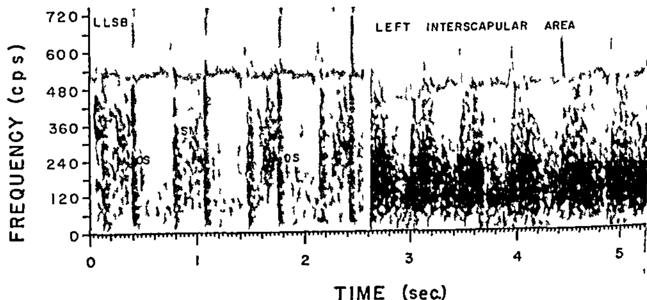


Fig 405 and 406 Mitral opening snap with coarctation

LLSB and interscapular area of back in P B (775818) 18 year old girl A murmur was present from birth Pulmonary capillary pressure was 18 mm Hg The left atrium was enlarged by x ray A diastolic murmur at the apex showed a presystolic crescendo In some other areas the abrupt and late onset of the systolic murmur a feature characteristic of coarctation was more clearly demonstrated The nondescript continuous low frequency noise is thought to be produced in arterial collaterals (left interscapular area)

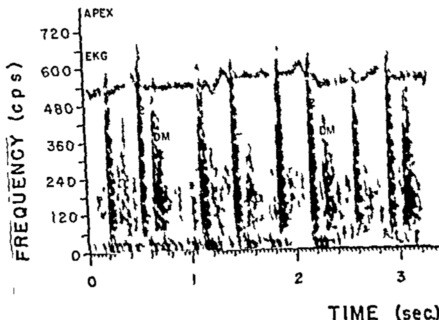


Fig 407 Coarctation with congenital mitral stenosis

Apex in R T (59 035) who in addition to the apical diastolic rumble had (by angiocardiography) enlargement of the left atrium and delay of the dye in the chamber

After operation there may be slight residual coarctation as indicated by a brachial artery pressure higher than femoral (1593) A systolic murmur may remain in such cases

PSEUDO COARCTATION There is a condition variously known as kinked aorta (188 154) buckled aorta atypical coarctation (1280) subclinical coarctation (1417) pseudo-coarctation (1437) in which there is distortion and often mild narrowing of the aortic arch at the level of the insertion of the ligamentum arteriosum On ordinary x ray of the chest the anomaly may simulate a tumor in that area The anomaly may occur in the Marfan syndrome and may be associated with aneurysm of an aortic sinus of Valsalva in patients with or without the Marfan syndrome Bruwer and Burchell (188) found a systolic murmur at the base of the heart in 9 of 10 cases and attributed it to turbulence of blood in the vicinity of the aortic kink In two of these patients the murmur had been detected in childhood and had been the basis for a diagnosis of organic heart disease DiGuglielmo and Guttadauro (354) reported a similar experience These clinical observations seem inconsistent with those of Gupta and Wiggers (617) who found that about 60 per cent constriction was necessary for murmur production in experimental coarctation

CONGENITAL PULMONARY REGURGITATION

ANATOMIC CONSIDERATIONS Isolated pulmonary regurgitation is an exceedingly rare congenital lesion At times the anatomic substrate is quadricuspid valve as mentioned previously In several of the clinical reports the anatomic basis for regurgitation has *not* been known It is difficult clinically to differentiate isolated pulmonary regurgitation on other also rare bases such as healed gonococcal endocarditis (1152)

Pulmonary regurgitation is occasionally *found* in association with other *congenital lesions* such as Eisenmenger's syndrome (in the case of Lavenne *et al* (855) there was total absence of pulmonary cusps) pulmonic stenosis and VSD etc Campeau *et al* (229) have also reported a case of total absence of pulmonary valve with VSD A *remarkable* feature was survival to the age of 32 years and performance of heavy work death was due to coronary occlusion

PHYSIOLOGIC CONSIDERATIONS On fluoroscopy there is likely to be striking hilar dance and by electrokymograms or roentgenkymograms large

has been demonstrated in association with coarctation (374), and a diastolic aortic murmur is likely to be present in such cases (560)

An important point is that when the aortic regurgitation associated with coarctation is of severe degree, as it sometimes is, the signs of coarctation may be submerged (for the unwary) The femoral pulses may feel quite adequate if it is not appreciated that the water hammer pulse present in the arms is not felt there and that there is in fact a delay in the femoral pulse

Wood (1590, p 335) states that one third of his patients with coarctation had an apical diastolic murmur suggesting mitral stenosis In this enumeration he excluded five patients with associated VSD or PDA which alone may be accompanied by such a murmur and also excluded three patients with rheumatic heart disease and mitral stenosis Cleland and his colleagues (272) found the same apical mid diastolic murmur without other evidences of mitral stenosis in 15 of 40 operated cases They were impressed with *its short duration, less rumbling quality and in*constant nature Of the 15 patients two had persistent ductus arteriosus and six had an early decrescendo diastolic murmur at the left sternal border Size of the left ventricle could not be related to presence or absence of the murmur in any convincing manner In six of the patients the mitral murmur was not heard during post operative observations However, this notation is of doubtful significance because of the inconstancy of the murmur before operation We have noted (see Figs 404, 405, 406) the occurrence of what appears to be an opening snap in at least two patients with coarctation (P B, 775818, K B 740310) The coarctation was of classic type in these patients, the left atrium was enlarged in the first, however

The same explanations for the diastolic murmur (Figs 404 and 407) are applicable as in the case of the same murmur occurring with congenital aortic stenosis dilatation of the ventricle with relative stenosis or fibroelastotic change (1162) in the mitral valve with actual obstruction Actual mitral stenosis has been described with coarctation as an associated congenital malformation (741 1156)

Because of the hypertension and the frequent presence of dilatation of the ascending aorta it is not surprising that an early systolic click is often heard in the aortic area

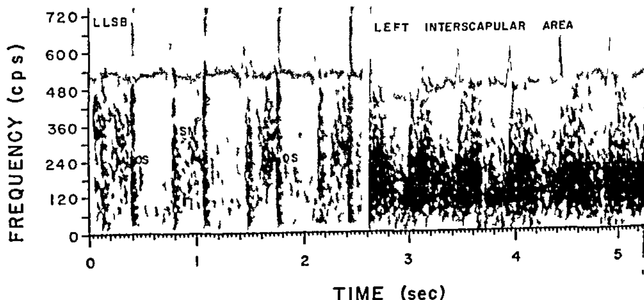


Fig 405 and 406 Mitral opening snap with coarctation

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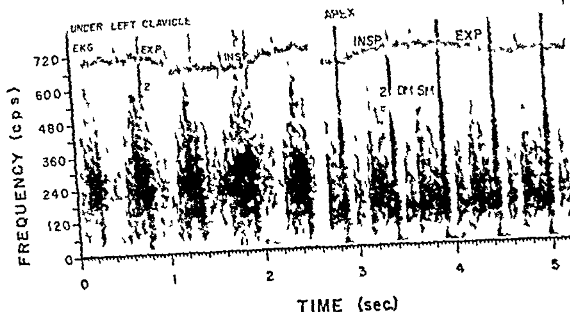


FIG. 410 Typical PDA

Left subclavicular area (*left*) and apex (*right*) in D. K. (764831), 5½ year-old female. The peak of intensity and frequency in the continuous murmur precedes S_2 by a significant interval. There is a mid diastolic murmur at the apex.

children particularly infants there is normally less aorto-pulmonary pressure differential. What difference in pressure exists may be greatest in systole. Kuhn and colleague (821) demonstrated no diastolic gradient in a patient with PDA and pulmonary hypertension who demonstrated only a systolic murmur. Rudolph and Mayer (1319) had a similar experience in a group of 20 infants (one year old or less) without continuous murmur. The aorto-pulmonary artery pressure gradient is closely related to murmur production in PDA.

When a PDA occurs proximal to coarctation of the aorta the shunt from aorta to pulmonary artery is exaggerated. When a PDA occurs distal to a coarctation or when pulmonary hypertension is present the direction of shunt is likely to be reversed at least during systole. A characteristic of the so-called reversed ductus is cyanosis in the feet and sometimes in the left arm without cyanosis in the other parts.

Patency of the ductus arteriosus can be demonstrated to persist for as long as fifteen hours after birth in the human by a dye method (1232), by differential arm-leg oxygen saturations (420) and by cineangiographic observation (950). Comparable demonstrations by use of the murmur have been reported by Dawes and colleagues (137, 338, 339) in newborn lambs in which a typical murmur of patent ductus is almost invariable. Why the murmur is not likewise the rule in newborn humans is discussed without definite conclusion by Born and his co-authors (137).

Physiologic factors which influence aorto-pulmonary pressure differential are likely to influence the volume of the shunt and the intensity of the murmur. Pulmonary hypertension has already been mentioned. Shepherd *et al.* (1384) were able to reverse a right-to-left shunt in a patient with pulmonary hypertension by having the patient breathe 99.6 per cent oxygen, a procedure known to reduce pulmonary arterial pressure.

It is interesting to reflect on the fact that with PDA the left ventricle labors under a diastolic overload whereas the right ventricle may, if there is pulmonary hypertension, be exposed to a systolic overload (1202). The diastolic overload is responsible for the fact that left ventricular ejection is prolonged relative to that of the right ventricle (821) and in turn for the paradoxical splitting of S_2 (587).

Bacterial endarteritis is one of the major risks

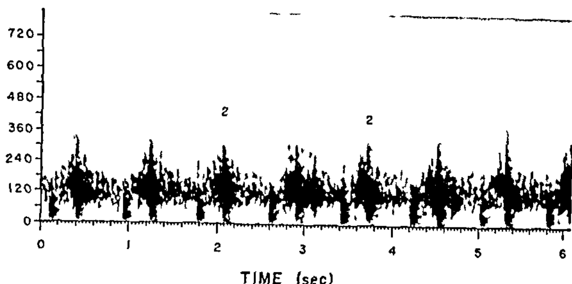


FIG 408 Patent ductus arteriosus

Typical continuous Gibson murmur with peak intensity and frequency in the vicinity of S—actually slightly before S Pulmonary area

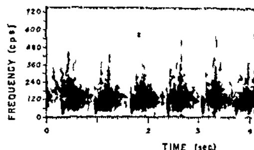


FIG 409 Patent ductus arteriosus

Typical Gibson murmur Pulmonary area

excursions of border movement in the main pulmonary artery with rapid rise and fall By cardiac catheterization there is usually a wide pulmonary arterial pulse pressure Particularly diagnostic is the presence of a sharp slope of the catacrotic limb with fall in pressure in diastole to a level almost *equal to that in the right ventricle (1117)* Using these features the clinical diagnosis of *isolated pulmonary regurgitation was made by* Kjellberg *et al* (800) Ford (469) and Morton and Stern (1117)

The clinical benignity of pulmonary regurgitation even absence of the pulmonary valve is consonant with the findings in animals with experimentally produced lesions of the pulmonary valve One cannot produce heart failure in dogs with only avulsion of the pulmonary cusps (52) It is likely that because of the low level of pressure in the pulmonary artery there is relatively little regurgitation

CARDIOVASCULAR SOUND The only feature of note is the early diastolic murmur which, in the absence of pulmonary hypertension, is likely to be low pitched and on the whole not conspicuous Holldack and Wolf (706) have noted a gap between S and the onset of the murmur in pulmonary *regurgitation Sometimes the murmur is* crescendo decrescendo rather than strictly de*crescendo from the second sound*

PATENT DUCTUS ARTERIOSUS (PDA)

(Syn Patent ductus botalli patent arterial duct)

ANATOMIC CONSIDERATIONS The usual site of the communication is between the aorta in the latter (or posterior) portion of the arch opposite the left subclavian ostium and the first part of the left main pulmonary artery A jet lesion may *occur in the intima of the pulmonary* artery opposite the opening of the ductus Occasionally, *in association with coarctation, the PDA com*municates with the aorta distal to the coarctation Associated aortic or subaortic stenosis has been emphasized recently by Mark and colleagues (1041)

PHYSIOLOGIC CONSIDERATIONS Because the pressure in the aorta is normally higher than *that* in the pulmonary artery at all times in the cardiac cycle the flow is at all times from aorta to pulmonary artery in uncomplicated cases of PDA The normal pressure gradient *between the aorta and* pulmonary artery is charted in Figure 101 In

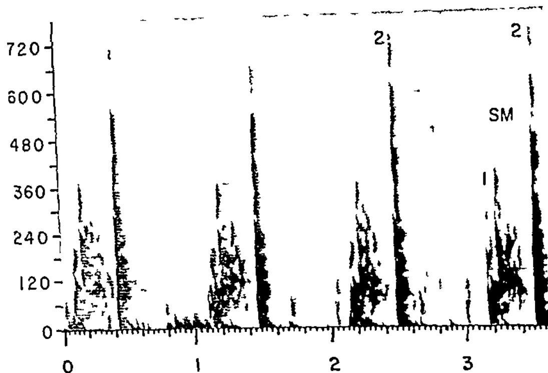


FIG. 419. Patent ductus arteriosus with pulmonary hypertension. Pulmonary area. Early systolic click initiates decrescendo systolic murmur. P2 greatly accentuated. No Gibson murmur.

whom the diagnosis of PDA was made by aortography. Keith and Forsyth (776) found a typical continuous murmur in three; in two a systolic murmur which at times extended into early diastole; in two no murmur at all; and finally in two a systolic murmur only located in the pulmonary area.

Kjellberg *et al.* (800) have pictured a continuous murmur which was recorded in an infant ten days old but which had disappeared completely by the time the child was four months old. A similar experience in two other patients was recorded. Presumably delayed but adequate closure of the ductus had occurred.

Rudolph and Mayer (1319) described 22 infants aged 1 to 12 months, only two of whom had a continuous murmur. The absence of the Gibson murmur could not be attributed to lack of shunt since in 20 pulmonary flow was about twice systemic flow. There was however no aorto-pulmonary pressure gradient during diastole. All these infants showed poor growth and development. Differences in the murmurs in various series may be the result of selection of different types of patients for study.

If the Gibson murmur is not quite continuous it is sometimes possible to demonstrate a gap of about 0.06 sec. between the first sound and the onset of the *murmur (1210)*.

Sometimes in children the continuous murmur is replaced by only a systolic murmur during attacks of some condition producing partial asphyxia (800). With the advent of left ventricular *failure or the elevation of pulmonary artery pressure* due to bronchopneumonia the continuous murmur is likely to disappear (227, 552) presumably because of elevation of pulmonary arterial pressure. I have known of *obliteration of a PDA* and disappearance of the characteristic murmur probably as a result of thrombosis accompanying bacterial endarteritis which was also cured spontaneously. Bishop (107) described a case in which closure is thought to have occurred between seven and fourteen years *also* possibly from endarteritis. Spontaneous cure of bacterial endarteritis (even without closure of the ductus) was described before the days of chemotherapy (257). Jager (712) observed obliteration from thrombosis occurring on marked atheromatosis in a 55 year old woman. In the case of Chiles and colleagues (260) a spon-

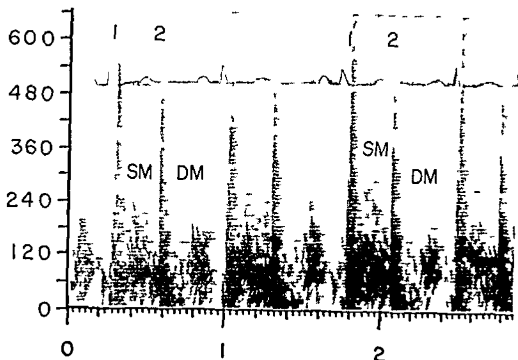


Fig 411 Patent ductus arteriosus
Mid diastolic rumble at the apex

in patent ductus arteriosus It occurred in 23 per cent of Abbott s cases (2) and in 28 6 per cent of the cases of Gelfman and Levine (538) Spontaneous closure of the ductus as a result of the thrombosis induced by bacterial infection has been reported (see below)

CARDIOVASCULAR SOUND The characteristic murmur called the Gibson murmur for George A Gibson (546) of Edinburgh (see p 29), is a continuous machinery murmur which has maximal intensity and frequency span in the vicinity of the second heart sound The murmur seems to be built around the second sound which it envelopes (638 1316) *It is usually loudest in the first and second left intercostal spaces under the left clavicle*

The reason for the shape of the Gibson murmur is not immediately evident since the peak of aorto pulmonary pressure differential (Fig 100) occurs earlier than the second heart sound where the peak of the murmur is usually situated As the pressure differential increases the size of the ductus may increase and there may be a resulting phase shift in the murmur Mannheimer (1033) states that the larger the ductus the earlier is the peak of the murmur In general close scrutiny of the Gibson murmur usually indicates that its peak is late systolic and not exactly coincident with S

Ziegler (1603) scrutinized the two following views which were held rather generally prior to his study

1 The usual time of appearance of the typical murmur is generally stated to be at the age of three to five years

2 Below this age the continuous murmur is supposedly infrequent and its presence justifies the suspicion of either the presence of a complicating defect such as pulmonary stenosis or coarctation of the aorta or an entirely different diagnosis than patent ductus arteriosus

In a series of 20 cases in individuals of an age three years or less Ziegler s findings (1603) were as follows

1 Except in the presence of a complicating pulmonary hypertension a typical continuous murmur was invariably present by the age of 18 months

2 A continuous murmur may be and frequently was present in infants with an uncomplicated patent ductus arteriosus as illustrated by two patients aged six weeks and five months respectively

In an addendum Ziegler referred to an infant five and one half and another eight months old each with continuous murmur Adler (9) heard a continuous murmur as early as six weeks *Holldack* and Wolf (706 p 128) present the phonocardiogram of an infant with PDA and a continuous murmur in the first days of life In nine infants in

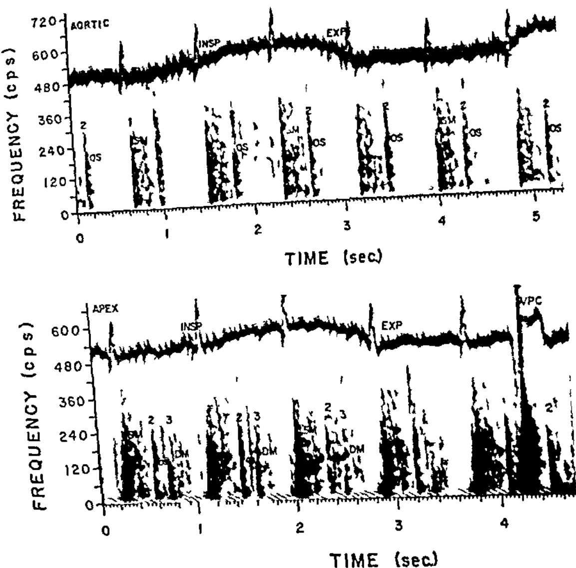


Fig. 414. Opening snap and diastolic rumble in P. T. (77331), a 20-year-old man with wide patent ductus arteriosus. Aortic (*above*) and apical (*below*) area. Both snap and rumble disappeared after ligation of PDA.

systolic right-to-left shunt (Fig 412) There may be a diastolic murmur of pulmonary regurgitation (see Table 16) Most reports (790 1000 1384 1381) of PDA in adults without typical murmur principally concern cases with pulmonary hypertension (1135) Holldack and colleagues (702) described a 16 year-old girl with a large ductus and essentially identical aortic and pulmonary pressures The only murmur was diastolic and was probably produced by pulmonary regurgitation The murmur was rough non-decrescendo and widely transmitted especially toward the left shoulder Even experienced observers confused it for a systolic murmur

Only rarely does a typical continuous murmur once established disappear When it does disappear as in a few reported cases (85 186) spontaneous closure is one poorly documented possibility (see p 401) and the development of pulmonary hypertension is another It is probable that most cases of PDA with pulmonary hypertension are that from birth Obviously the wide PDA (see below) is likewise that from birth

A PDA entering the aorta at or beyond a

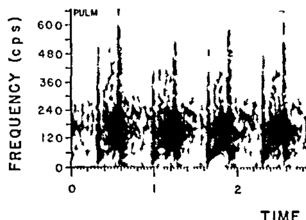


FIG 413 Musicality in murmur of PDA
S H (352841) 18 years old shows a continuous murmur typical of PDA except for the evidence of musicality in diastole

taneous closure was discovered to have occurred 15 years after the onset of subacute bacterial endarteritis The infection had been treated with a sulfonamide in several courses

Soulié and his colleagues (1418) by means of a balloon catheter introduced into the aorta by the usual method of right heart catheterization, were able to occlude a ductus arteriosus and abolish the continuous murmur Failure of the murmur to disappear was proposed as a test for associated cardiovascular malformation

There is often in the pulmonary area an early systolic click related to the dilated pulmonary artery

At the apex there is likely to be a mid diastolic Carey Coombs type of murmur when the volume of the aorto pulmonary shunt is large (Figs 410 411 and 414) Rayam and Darley (1248) found such a murmur in 9 of 21 patients Actual mitral stenosis due to fetal fibroelastosis may occur with patent ductus arteriosus (38 145 224 682 1162) The differentiation of functional from organic mitral stenosis may be difficult Left atrial enlargement may occur in both In organic stenosis an opening snap and a presystolic element of the murmur at slow heart rates are more likely to occur and pulmonary capillary pressure at right heart catheterization is likely to be higher

Paradoxical splitting of S_2 was found by Gray (587) in 10 of 29 cases of PDA the aortic component followed the pulmonary component and splitting was greatest in expiration This splitting is thought to be related to the discrepant stroke volumes of the ventricles, that of the left ventricle being larger Systole in the left ventricle is prolonged in relation to the right ventricle Splitting is *not* present more often because of the partial cancelling effect of the normal aortic pulmonary closure sequence

With a large patent ductus arteriosus there may be Duroziez's sign In Routier's 14 cases, the sign was present in eight Other peripheral signs usually associated with AR may also be present

ATYPICAL PDA In the "*reversed ductus*" syndrome—patent ductus arteriosus with pulmonary hypertension and reversed shunt—there may be no murmur or only a systolic murmur related either to the dilated pulmonary artery or to the

TABLE 16
*Murmurs in selected patients with balanced or reversed PDA proved by oxygen saturation studies or other studies**

Author	Patient	Age	Murmurs
	No	Years	
Cosh (1953)	1	27	Faint pulmonary systolic
	2	9	Loud systolic over precordium
Hultgren *et al* (1953)	2	34	No murmur
	3	31	Systolic LLSB
	4	21	Systolic LLSB
Dammann *et al* (1953)	2	29	Pulmonary systolic and thrill diastolic at LSB
	3	20	Systolic LSB
	4	30	Pulmonary systolic and diastolic
Smith (1954)	—	19	Systolic third left interspace
Emslie Smith (1955)	—	10	Loud systolic maximal in pulmonary area
Whitaker *et al* (1955)	2	26	No murmur
	3	6	Pulmonary systolic
	4	58	No murmur
	5	22	Pulmonary diastolic
	6	46	No murmur
	7	8	Pulmonary systolic and diastolic
	8	34	Pulmonary systolic
Campbell (1955)	28	19	Pulmonary systolic
	29	19	Rough pulmonary systolic faint pulmonary diastolic
	30	40	Loud pulmonary diastolic
	32	27	Soft pulmonary systolic

* Adapted from Born *et al* (137) with additions

CONDITIONS WITH AUSCULTATORY SIMULATION OF PDA

Probably as a generalization the statement made by Muir and Brown (1131) in 1935 is true that in its typical form the Gibson murmur is the most pathognomonic of all murmurs. Yet as experience increases the list of simulating conditions is enlarged.

Any intrathoracic arteriovenous fistula (PDA is essentially such) may be accompanied by a machinery murmur of the Gibson type (546) (Fig. 416, 417). Examples are aortic septal defect or aorto-pulmonary window (see later) and rupture of an aneurysm of a sinus of Valsalva into the pulmonary artery, right ventricle or right atrium. Also the artificial ductus created by the Blalock-Taussig and Potts operation produces a murmur of this character.

Fistulous communication between a coronary artery and the coronary vein (1189) or sinus (331), a chamber of the heart (762) or the pulmonary artery (105) may be accompanied by a Gibson murmur (Fig. 418). Sometimes the murmur is predominantly diastolic or has a diastolic, not a late systolic, accentuation caused by the fact that coronary flow is maximal during diastole. Knoblich and Rawson (806) reported the case of a 53 year old businessman whose murmur, discovered at age 18, was described as "a peculiar harsh Grade III diastolic apical murmur of blowing character." The location of the murmur will help differentiate coronary AV fistula from PDA in those cases in which it is maximal over the lower precordium or apex. However, cases occur in which the murmur is located high on the left (1506). Davidson and colleagues (331) reported a 28 year-old patient in whom a huge communication between the left circumflex coronary artery and the coronary sinus resulted in a loud continuous murmur in the pulmonary area, wide pulse pressure, and high output cardiac failure. The superficial quality of the murmur may suggest that coronary AV fistula is responsible. (The oxygen content of coronary sinus blood ordinarily very low, is likely to be high in cases of coronary AV fistula. However, if the communication is with the right ventricle or pulmonary artery, the oxygen step-up at cardiac catheterization will be found at these sites.) Recently Edwards (110A) and others (241B) have resurrected the old observation (184A) that flow in an anomalous right coronary artery arising from the pulmonary artery is *toward* the pulmonary artery. The development of an arterio-arterial fistula which functionally is an arteriovenous fistula may ensue.

The AV fistula may be in the thoracic cage—internal mammary (1083, 1555) or intercostal—and be either congenital or the result of rib fracture or stab wound.

Arteriovenous fistulas which are fundamentally congenital may not enlarge to the point that clinical manifestations are produced until rather late in life (893): congenital AV fistulas are in many instances progressive, not static, lesions. The progressive character is noted in connection with AV fistulas in the extremities, in the lungs and at most other sites.

In many, but not all, cases of *pulmonary arteriovenous fistulas* a murmur may be audible in overlying areas of the chest (1513). It is likely to be a continuous murmur but may be loud in inspiration and almost inaudible with expiration (1127). Other writers (706, p. 142) describe the murmur as being least loud in inspiration. Judging by the reports in the literature one (1101) can conclude that about two thirds of patients show a murmur directly attributable to the fistula(s). The *shunt* of blood is occasionally of sufficient volume to result in a functional mitral diastolic murmur. The characteristic syndrome includes cyanosis, polycythemia and clubbing of the fingers. Heart failure does not occur commonly, contrary to the situation with peripheral AV fistula, probably because a fistula large enough to cause heart failure would kill sooner by its effect on arterial oxygen saturation. The patient may get into one or more of three main types of serious trouble: (1) hemoptyses, which may be fatal; (2) infection, that is, bacterial endarteritis on the fistula; (3) neurologic complications—thromboses secondary to the polycythemia and brain abscess which occurs with increased incidence in this as in other varieties of right to left shunt. The fact that about one half of all cases of the pulmonary AV fistula occur as part of the Osler-Rendu-Weber syndrome (1521) is indicated

See references 553 and 1105.

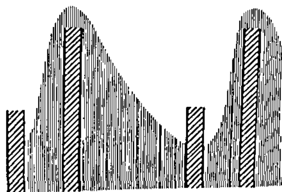
coarctation is likely to be a "reverse ductus." The auscultatory findings are atypical and do not permit diagnosis by this means alone.

The second major category of atypical PDA is what Aitken in his excellent study (14) refers to as "the *wide patent ductus arteriosus*." There may be moderate pulmonary hypertension, but what hypertension is present is more the effect of high flow through the pulmonary circuit—the volume of pulmonary flow in these cases may be at least three times that of systemic flow. The clinical features Aitken emphasizes are (1) the presence of a suprasternal thrill which often is also present in the neck vessels, and (2) the replacement of the typical Gibson murmur by a systolic-diastolic murmur resembling that of aortic stenosis and regurgitation. The patients usually demonstrate left atrial enlargement and an apical diastolic rumble. Even a mitral opening snap may occur in these cases in the absence of organic change in the valve (Fig. 414). Neill and Mounsey (1140A) had five patients with PDA with opening snap. In all but one the snap disappeared after operation. Elevation of pulmonary capillary pressure (to an average of 19.6 mm. Hg in 14 patients) agreed well with these other signs of relative mitral stenosis. Collapsing peripheral pulse and wide pulse pressure was more likely to be present. Early severe disability is usual. Mannheimer and Sandblom (1036) and Dammann and Sell (328) together have described 21 cases of wide ductus and also emphasize that the murmur is likely to be systolic-diastolic or only systolic. The thrill in the neck vessels and suprasternal notch is probably produced as a result of the high flow through the aorta. Pertinent to the absence of the typical Gibson murmur may be the characteristic pressures recorded at cardiac catheterization. In the region of the pulmonary artery near the orifice of the ductus the high pressure of the aorta is transmitted to the catheter, whereas both proximal and distal to that point pressures are lower. Several authors (1486) have emphasized the "mitral syndrome" or "pseudo-mitral syndrome" which tends to be unusually striking in the patient with a large ductus: apical rumble, enlarged left atrium, left atrial hypertrophy by electrocardiogram, elevated pulmonary capillary pressure, gradient across the mitral valve demonstrated at operation.

Wide PDA can be difficult to differentiate from aortic septal defect.

THORACIC A-V FISTULA

(e.g. patent ductus arteriosus, thyroid bruit, Blalock-Taussig anastomosis, pulmonary, coronary and other intrathoracic A-V fistulae)



AORTIC STENOSIS AND REGURGITATION

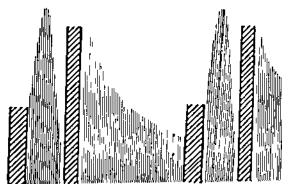


FIG. 415. Comparison of the frequency-time pattern of the murmur of thoracic arteriovenous fistula with that of aortic stenosis and regurgitation. (The Christmas tree murmur of aortic stenosis should have been indicated as stopping with a brief gap before S_2.)



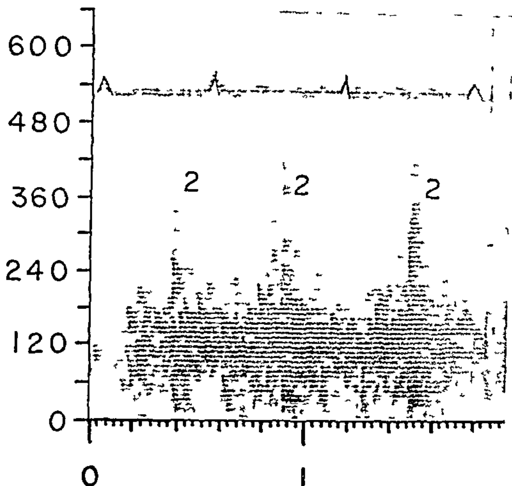
Fig 418A and 419B Suspected arteriovenous fistula of the coronary circulation
A continuous murmur was audible over the lower sternum at the level of the fifth intercostal space The patient a 10 year-old boy had no symptoms (Fig 418) Anteroposterior and (Fig 419) lateral view exposed 6 sec after the injection of contrast medium At the time of aortic opacification a dilated tortuous vessel (arrows) was filled (Courtesy of Cooley and Sloan (901))

by family history or the presence of telangiectases of the skin and mucous membrane can be used in diagnosis telangiectasis should be sought on the lips and elsewhere and the family examined

Claiborne and Hopkins (266) describe a case with PDA like murmur in which an AV fistula in the lung represented a communication between a branch of the aorta and the pulmonary vessel rather than pulmonary artery and pulmonary vein See Fig 214 for a similar case of mine

With *defect of the aorto-pulmonary septum* (289 323 352 529, 549 555 613 811) the peripheral signs of PDA such as wide pulse pressure usually are present The murmur is usually said to be situated lower than that of PDA being maximal right over the sternum at the level of the second and third intercostal spaces or at the left sternal border in this area However this differentiating point has been questioned (39) The murmur has been continuous in a few patients in some it has extended into diastole but not all the way through and in some it has been confined to systole (Fig 420) Pulmonary hypertension and pulmonary regurgitation is probably more likely to occur in these cases than in PDA In a review of 21 cases reported in the literature one group of writers found a continuous murmur in only 2 (9 per cent) Atypical murmurs seem typical of aortic septal defect In one case in 11 year-old child operated on by Bailey (39) it was impossible to close the proximal portion of the defect completely because of risk of embarrassing coronary circulation In spite of incomplete closure pulmonary hypertension was reduced and the murmur only systolic before operation became systo-diastolic and later continuous after operation

In *pseudotruncus arteriosus*—pulmonary atresia with blood supply to the lungs by the bronchial arteries—a continuous murmur with the pattern of that of PDA is produced in the dilated bronchial arteries which are carrying a relatively large volume of blood (316) The explanation for the continuous nature of the murmur probably is related to the low pressure in the pulmonary circuit—a special case of the phenomenon described by Myers Murdaugh McIntosh and Blaisdell (1136) (see p 232) In true truncus arteriosus communis when there is large pulmonary flow (288) the findings suggest PDA This syndrome was found in four of 400 children coming to





Figs 416 and 417 Congenital thoracic arteriovenous fistula

(*Above*) Gibson murmur heard and recorded in third right interspace of 50 year old female (V J 145562) followed in this hospital for 19 years *The murmur and cardiomegaly have been present* throughout but both seem to have been increasing slowly through the years She has been virtually asymptomatic and pulse pressure is normal The diagnosis has been aortic stenosis and regurgitation With the passage of years right axis deviation has appeared and in recent months atrial *fibrillation These developments are not surprising in light* of the pronounced dilatation of the right atrium and ventricle on x rays (*below*) including angiocardiogram (*lower right*) The vessels involved in the fistula are unknown a coronary artery to coronary sinus communication is considered likely A patient (C Y 258876 age 67) with strikingly *similar story and identical sound recording* has been observed at this hospital since 1915

tenosis of the right pulmonary artery (960) is probably closely akin. In this condition the main pulmonary artery is affected. In one series of five cases PDA was also present in four. A grade III or IV systolic murmur at the base of the heart was described. When the systolic murmur, especially when accompanied by a thrill, is maximal at the right of the sternum, aortic stenosis is simulated (1108A).

In the *aortic arch syndromes* (1312)—diminished or absent pulses in arteries arising from the arch of the aorta—a continuous murmur (746) may be heard at the base of the neck, either on the right (550, 905) or on the left (6, 543, 1136, 1394, 1423). Particularly in the latter case is PDA simulated. The murmur is not well heard below the clavicle however. Murmurs are heard over the back (503, 1394) generated in collaterals in this condition which has been appropriately termed reversed coarctation. However, the principle outlined by Myer and colleagues (1176) is probably responsible in most cases for the murmur at the base of the neck.

I have been told (961) of a case of *dissected aneurysm of the aorta* with a continuous murmur supposedly identical to that of PDA; aortic regurgitation was not present. It may be that this was a special case of the aortic arch syndrome with continuous murmur or the murmur may have been generated in some less well understood manner in the complicated distortion of the aorta.

Venous hums may be very loud in children and readily audible over the upper part of the thorax. Obliteration by pressure on the neck veins and accentuation or attenuation by appropriate measures (see p. 226) usually suffices in identifying the nature of the sound. Furthermore venous hum is usually most intense in diastole and at the right of the sternum if audible at all over the upper chest.

A continuous murmur precisely like that of PDA except for location under the right clavicle has been described in cases of *total anomalous pulmonary venous return* of the figure-of-eight or snowman type (see p. 3[illegible]).

Usually the pattern of murmurs in AS and AP is quite distinct from that in PDA (Fig. 41.5). I have seen cases of *aortic stenosis and regurgitation* in which there is a continuous murmur much like

TABLE 5

Conditions with auscultatory simulation of patent ductus arteriosus

1 Other intrathoracic AV fistulas
  a Aortic septal defect
  b Rupture of sinus of Valsalva (syphilitic (670) or congenital (913, 1164) aneurysm, SBE)
  c Artificial (surgical) ductus
  d Coronary artery (335, 888, [illegible], 235) to coronary vein (1189) or heart chamber (1140A) or pulmonary artery (10[illegible]) (67A)
  e AV fistula in thoracic cage
    congenital, e.g., internal mammary (10[illegible], 1528)
    traumatic, e.g., after rib fracture
  f Intrapulmonary AV fistula, pulmonary artery to pulmonary vein, aortic branch to peripheral pulmonary artery (3[illegible])
  g Subclavian (750)
  h Rupture of syphilitic aneurysm of the aorta into the pulmonary artery ([illegible]) or superior vena cava ([illegible])
2 Ventricular septal defect
  a With aortic regurgitation
  b With pulmonary regurgitation (13[illegible])
3 Systemic collaterals in coarctation of the aorta
4 Bronchial collaterals to lung in pulmonary atresia or severe pulmonary stenosis (pseudotruncus arteriosus)
5 Syndrome of true truncus arteriosus communis with large pulmonary flow (258)
6 Aortic arch syndrome (131[illegible])
  Multiple congenital stenoses of pulmonary arteries (29, 47, 671A, 1109, 15[illegible])
8 Venous hum
9 Mammary souffle
10 Total anomalous pulmonary venous return ([illegible])
11 Aortic stenosis and regurgitation

that of PDA over the lower sternum and xiphoid. Graphically the murmurs in that area have the same pattern as that of PDA although in the pulmonary and aortic areas the appearance and sound is quite typical of double to and fro murmur. In my experience about one in each one hundred cases of aortic stenosis and regurgitation displays this phenomenon. The mechanism is not clear. Of course the association of a lesion such as Chiari's network of the right atrium cannot be excluded. Such a lesion can generate a continuous murmur of this type (p. 410).

In late pregnancy and the early parturient state a continuous murmur resembling the Gibson murmur is audible over the upper margins of the

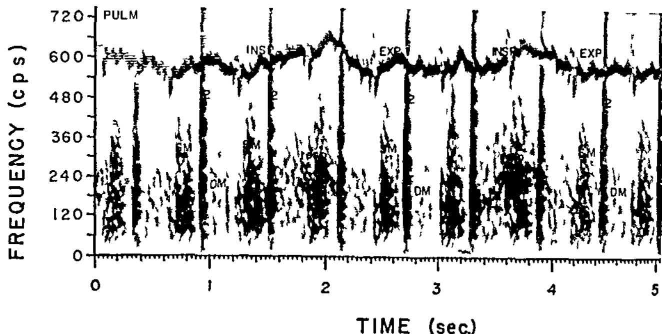


FIG 420 Aortic septal defect

C K (76156) 7 year old boy had always had only a systolic murmur at the left sternal border There was an apical mid diastolic murmur VSD was the diagnosis before cardiac catheterization which revealed an oxygen step up in the pulmonary artery The pressure in the pulmonary artery was 90/51 mm Hg and that in the femoral artery about the same Yet it was calculated that pulmonary blood flow was about 2½ times systemic flow At operation (performed by Dr Henry T Bahnson) the anticipated ductus was not discovered but rather a large aortic septal *defect measuring about 18 mm in diameter and larger probably than either the aorta or pulmonary artery* It was possible to close the defect completely Pulmonary artery pressure was normal after the procedure and the patient has done well

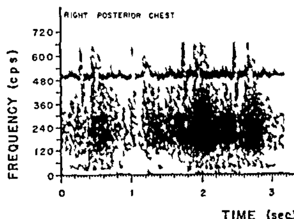


FIG 421 Continuous murmur of bronchial collaterals *in tetralogy of Fallot*

A 28 year old white male (772277) was clearly demonstrated—by angiocardiography and cardiac catheterization—to have tetralogy of Fallot Over almost all the right lung field there was a loud continuous murmur identical in quality and timing to that of PDA Because of clubbing polycythemia and cyanosis with relatively little incapacitation multiple pulmonary arteriovenous fistulas were at first diagnosed but the final conclusion was that bronchial collaterals are responsible for the PDA like murmur

surgery with the diagnosis of PDA (288) One had a murmur in systole only, the other three had a rather rumbling murmur located in the second and third left interspaces and extending well into but not through diastole

Rare cases of *multiple congenital stenosis of medium sized pulmonary arteries* have been reported to have a Gibson type of murmur (29 422 616 1109 1564) Again, that the murmur in this condition is continuous not merely systolic is explained by the work of Myers *et al* (1136) Eldridge Selzer, and Hultgren (422) described three cases in two of whom a continuous murmur was heard These two patients were demonstrated by cardiac catheterization to have a difference in pressure proximal and distal to the stricture during all parts of the cardiac cycle The case with only a systolic murmur demonstrated little pressure difference during ventricular diastole Powell and Hiller (1231) reported "a long almost continuous murmur at the pulmonary area" in a case of pulmonary coarctation' The syndrome of

stenosis of the right pulmonary artery (960) is probably closely akin. In this condition the main pulmonary artery is affected. In one series of five cases PDA was also present in four. A grade III or IV systolic murmur at the base of the heart was described. When the systolic murmur, especially when accompanied by a thrill, is maximal at the right of the sternum, aortic stenosis is simulated (1108A).

In the *aortic arch syndromes* (1312)—diminished or absent pulses in arteries arising from the arch of the aorta—a continuous murmur (746) may be heard at the base of the neck, either on the right (550, 905) or on the left (6, 543, 1176, 1394, 1423). Particularly in the latter case is PDA simulated. The murmur is not well heard below the clavicle, however. Murmurs are heard over the back (503, 1394) generated in collaterals in this condition, which has been appropriately termed reversed coarctation. However, the principle outlined by Myer and colleagues (1136) is probably responsible in most cases for the murmur at the base of the neck.

I have been told (961) of a case of *dissected aneurysm of the aorta* with a continuous murmur supposedly identical to that of PDA. Aortic regurgitation was not present. It may be that this was a special case of the aortic arch syndrome with continuous murmur, or the murmur may have been generated in some less well understood manner in the complicated distortion of the aorta.

Venous hums may be very loud in children and readily audible over the upper part of the thorax. Obliteration by pressure on the neck veins and accentuation or attenuation by appropriate measures (see p. 226) usually suffices in identifying *the nature of* the sound. Furthermore, venous hum is usually most *intense in diastole* and at the right of the sternum *if audible* at all over the upper chest.

A continuous murmur precisely like that of PDA except for location under *the right clavicle* has been described in cases of *total anomalous pulmonary venous return* of the "figure of eight" or "snowman" type (see p. 353).

Usually the pattern of murmurs in AS and AR is quite distinct from that in PDA (Fig. 415). I have seen cases of *aortic stenosis and regurgitation* in which there is a continuous murmur much like that of PDA over the lower sternum and xiphoid. Graphically the murmurs in these areas have the same pattern as that of PDA, although in the pulmonary and aortic *areas the* appearance and sound is quite typical of double to-and-fro murmur. In my experience about one in each one hundred cases of aortic stenosis and regurgitation displays this phenomenon. The mechanism is not clear. Of course, the association of a lesion such as Chiari's network of the right atrium cannot be excluded. Such a lesion can generate a continuous *murmur of this type* (p. 410).

In late pregnancy and the early parturient state a continuous murmur *resembling the Gibson murmur is audible* over the upper margins of the

TABLE 17

Conditions with auscultatory simulation of patent ductus arteriosus

1. Other intrathoracic AV fistulas
   a. Aortic septal defect
   b. Rupture of sinus of Valsalva (syphilitic ([illegible]) or congenital (913, 1164) aneurysm, SBE)
   c. Artificial (surgical) ductus
   d. Coronary artery ([illegible], 888, p. 250) to coronary vein (1181), to heart chamber (1140A), or pulmonary artery (1065) ([illegible])
   e. AV fistula in thoracic cage
      congenital, e.g., internal mammary ([illegible], 1528)
      traumatic, e.g., after rib fracture
   f. Intrapulmonary AV fistula: pulmonary artery to pulmonary vein, aortic branch to peripheral pulmonary artery ([illegible])
   g. Subclavian ([illegible])
   h. Rupture of syphilitic aneurysm of the aorta into the pulmonary artery ([illegible]) or superior vena cava ([illegible])
2. Ventricular septal defect
   a. With aortic regurgitation
   b. With pulmonary regurgitation (1332)
3. Systemic collateral in coarctation of the aorta
4. Bronchial collaterals to lung in pulmonary atresia or severe pulmonary stenosis ([illegible])
5. Syndrome of true truncus arteriosus communis with large pulmonary flow (255)
6. Aortic arch syndromes (1312)
7. Multiple congenital stenoses of pulmonary arteries ([illegible], 1109, [illegible])
8. Venous hum
9. Mammary souffle
10. Total anomalous pulmonary venous return ([illegible])
11. Aortic stenosis and regurgitation

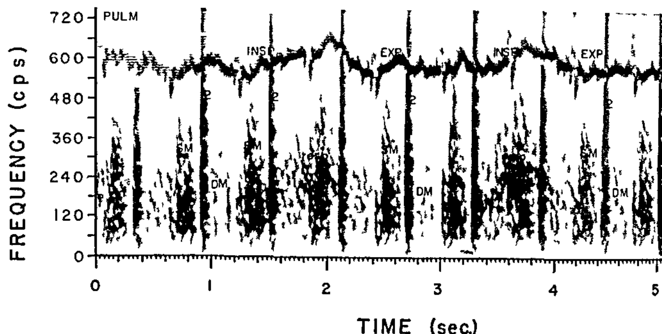


Fig. 420 Aortic septal defect

C K (76456) 7 year old boy had always had only a systolic murmur at the left sternal border There was an apical mid diastolic murmur VSD was the diagnosis before cardiac catheterization which revealed an oxygen step up in the pulmonary artery The pressure in the pulmonary artery was 90/54 mm Hg and that in the femoral artery about the same Yet it was calculated that pulmonary blood flow was about 2½ times systemic flow At operation (performed by Dr Henry T Bahnson) the anticipated ductus was not discovered but rather a large aortic septal defect measuring about 18 mm in diameter and larger probably than either the aorta or pulmonary artery It was possible to close the defect completely Pulmonary artery pressure was normal after the procedure and the patient has done well

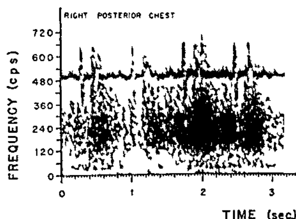


Fig 421 Continuous murmur of bronchial collaterals in tetralogy of Fallot

A 28 year old white male (772277) was clearly demonstrated—by angiocardiography and cardiac catheterization—to have tetralogy of Fallot Over almost all the right lung field there was a loud continuous murmur identical in quality and timing to that of PDA Because of clubbing polycythemia and cyanosis with relatively little incapacitation multiple pulmonary arteriovenous fistulas were at first diagnosed but the *final conclusion* was that bronchial collaterals are responsible for the PDA like murmur

surgery with the diagnosis of PDA (288) One had a murmur in systole only, the other three had a rather rumbling murmur located in the second and third left interspaces and extending well into but not through diastole

Rare cases of *multiple congenital stenosis of medium sized pulmonary arteries* have been reported to have a Gibson type of murmur (29, 422, 616, 1109 1564) Again that the murmur in this condition is continuous not merely systolic, is explained by the work of Myers *et al* (1136) Eldridge Selzer and Hultgren (422) described three cases in two of whom a continuous murmur was heard These two patients were demonstrated by cardiac catheterization to have a difference in pressure proximal and distal to the stricture during all parts of the cardiac cycle The case with only a systolic murmur demonstrated little pressure difference during ventricular diastole Powell and Hiller (1231) reported "a long almost continuous murmur at the pulmonary area" in a case of pulmonary coarctation" The syndrome of

tenosis of the right pulmonary artery (1101) is probably closely akin. In this condition the main pulmonary artery is affected. In one series of five cases PDA was also present in [illegible]. A grade III or IV systolic murmur at the base of the heart was described. When the systolic murmur, especially when accompanied by a thrill, is maximal at the right of the sternum aortic stenosis is imitated (1108A).

In the *aortic arch syndromes* (1712)—diminished or absent pulses in arteries arising from the arch of the aorta—a continuous murmur (1461) may be heard at the base of the neck, either on the right (1401, 901) or on the left (1447, 1171, 1794, 1422). Particularly in the latter case is PDA simulated. The murmur is not well heard below the clavicle, however. Murmurs are heard over the [illegible] (1401) generated in collaterals in this condition which has been appropriately termed reversed coarctation. However, the principle outlined by Myer and colleagues (1171) probably responsible in most cases for the murmur at the base of the neck.

I have been told (461) of a case of *dissecting aneurysm of the aorta* with a continuous murmur supposedly identical to that of PDA; aortic regurgitation was not present. It may be that this was a variant case of the aortic arch syndrome [illegible] continuous murmur or the murmur may have been generated in some less well understood manner in the complicated [illegible] of the aorta.

[illegible] hums may be very loud in children and [illegible] audible over the upper part of the thorax. [illegible] by pressure on the neck veins and [illegible] or attenuation by appropriate [illegible] usually affords a distinguishing [illegible] of the sound. Furthermore venous [illegible] usually not intense in diastole and at the right of the sternum it audible [illegible] the upper chest.

[illegible] murmur precisely like that of [illegible] except for location under the right clavicle has been described in cases of *total anomalous pulmonary venous return* of the [illegible] man type (see p. [illegible]).

[illegible] the pattern of murmurs in [illegible] and [illegible] from that in PDA (Fig. 41). I have seen cases [illegible] and [illegible] in which there is a continuous murmur much like

TABLE [illegible]

[illegible]

1. [illegible]
   a. [illegible]
   b. [illegible]
   c. [illegible]
   d. Coronary artery [illegible]
   e. [illegible]
   f. [illegible] pulmonary artery [illegible]
   g. [illegible]
   h. [illegible] the pulmonary artery [illegible]
2. [illegible]
   a. [illegible]
   b. [illegible]
3. [illegible]
4. [illegible]
5. [illegible] large pulmonary flow [illegible]
6. [illegible]
7. Multiple [illegible] of pulmonary arteries [illegible]
8. [illegible]
9. Mammary souffle
10. Total anomalous pulmonary venous return
11. [illegible]

that of PDA [illegible]. Graphically the murmurs in [illegible] have the same pattern as that of PDA, although [illegible] the pulmonary and aortic areas [illegible] and sound [illegible] typical [illegible] murmur. In my experience [illegible] in each [illegible] hundred cases of [illegible] display this phenomenon. The [illegible]. Of course [illegible] a Chiari network of the right atrium [illegible] excluded, such a [illegible] can generate a [illegible] murmur of this type (p. 410).

In late pregnancy and the early puerperium a continuous murmur resembling the [illegible] murmur is audible [illegible] the upper margins of the

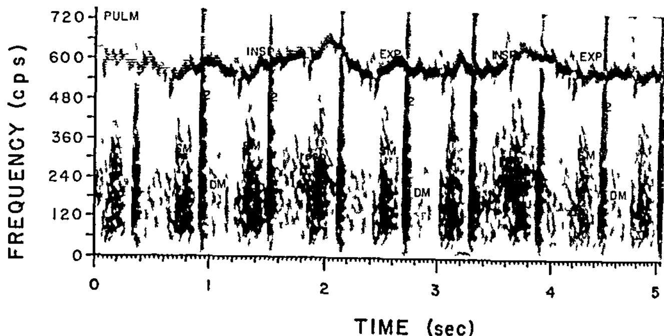


FIG 420 Aortic septal defect

C K (76406) 7 year old boy had always had only a systolic murmur at the left sternal border There was an apical mid diastolic murmur VSD was the diagnosis before cardiac catheterization which revealed an oxygen step up in the pulmonary artery The pressure in the pulmonary artery was 90/54 mm Hg and that in the femoral artery about the same Yet it was calculated that pulmonary blood flow was about 2½ times systemic flow At operation (performed by Dr Henry T Bahnson) the anticipated ductus was not discovered but rather a large aortic septal defect measuring about 18 mm in diameter and larger probably than either the aorta or pulmonary artery It was possible to close the defect completely Pulmonary artery pressure was normal after the procedure and the patient has done well

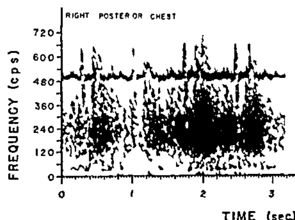


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In the *aortic arch syndrome* (1312)—diminished or absent pulses in arteries arising from the arch of the aorta—a continuous murmur (746) may be heard at the base of the neck, either on the right (550, 905) or on the left (6, 543, 1179, 1394, 1422). Particularly in the latter case is PDA simulated. The murmur is not well heard below the clavicle, however. Murmurs are heard over the back (550, 1394) generated in collaterals in this condition, which has been appropriately termed reversed coarctation. However, the principle outlined by Myer and colleagues (1136) is probably responsible in most cases for the murmur at the base of the neck.

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Venous hums may be very loud in children and readily audible over the upper part of the thorax. Obliteration by pressure on the neck veins and accentuation or attenuation by appropriate measures (see p. 220) usually suffices in identifying the nature of the sound. Furthermore, venous hum is usually most intense in diastole and at the right of the sternum, if audible at all over the upper chest.

A continuous murmur precisely like that of PDA except for location under the right clavicle has been described in cases of *total anomalous pulmonary venous return* of the "figure-of-eight" or "snowman" type (see p. 353).

Usually the pattern of murmurs in AS and AR is quite distinct from that in PDA (Fig. 115). I have seen cases of *aortic stenosis and regurgitation* in which there is a *continuous* murmur much like

TABLE 17

Conditions with auscultatory simulation of patent ductus arteriosus

1. Other intrathoracic AV fistula
   a. Aortic septal defect
   b. Rupture of sinus of Valsalva (syphilitic (6) or congenital (911, 1164) aneurysm, SBE)
   c. Artificial (surgical) ductus
   d. Coronary artery (335, 888, p. 265) to coronary vein (1187), or heart chamber (1140A), or pulmonary artery (105) (6 A)
   e. AV fistula in thoracic cage
      congenital, e.g., internal mammary (1025, 1528)
      traumatic, e.g., after rib fracture
   f. Intrapulmonary AV fistula, pulmonary artery to pulmonary vein, aortic branch to peripheral pulmonary artery (38)
   g. Subclavian (60)
   h. Rupture of syphilitic aneurysm of the aorta into the pulmonary artery (316, 125 , 1513) or superior vena cava (1 B, 4 , 1 0)
2. Ventricular septal defect
   a. With aortic regurgitation
   b. With pulmonary regurgitation (13)
3. Systemic collaterals in coarctation of the aorta
4. Bronchial collaterals to lung in pulmonary atresia or severe pulmonary stenosis (pseudotruncus arteriosus)
5. Syndrome of true truncus arteriosus communis with large pulmonary flow (788)
6. Aortic arch syndrome (1312)
7. Multiple congenital stenoses of pulmonary arteries (4 , 671A, 1109, 1563)
8. Venous hum
9. Mammary souffle
10. Total anomalous pulmonary venous *return* (   )
11. Aortic stenosis and regurgitation

that of PDA *over* the lower sternum and xiphoid. Graphically, the murmur in that area have the same pattern as that of PDA, although in the pulmonary and aortic area the appearance and sound is quite typical of *double* to and fro murmur. In my experience about one in each one hundred cases of aortic stenosis and regurgitation displays this *phenomenon*. The mechanism is not clear. Of course, the association of a lesion such as Chiari's network of the right atrium cannot be excluded. Such a lesion can generate a continuous murmur of this type (p. 110).

In late pregnancy and the early parturient state a continuous murmur resembling the Gibson murmur is audible over the upper margins of the

congested breasts—"the *mammary souffle*." Because of its accentuation in late systole it appears to be arterial, not venous. It has a superficial quality and its superficial origin is further supported by the fact that it can be obliterated by pressure with the bell of the stethoscope. There is a fickleness about the murmur such that it appears and disappears more or less unaccountably. An intermediate grade of pressure with the stethoscope bell can intensify the murmur or cause one not previously present to appear. The murmur may have a musical quality. Knowledge of the mammary souffle (p. 233) and of the characteristics mentioned is sufficient to distinguish this phenomenon from PDA.

The syndrome of VSD with AR may simulate PDA; patients have been mistakenly operated on for presumed PDA (e.g., T. R., 726671, 26 year old white female). As suggested previously (p. 362), it is possible that the murmur of the VSD-AR syndrome and that of ruptured sinus of Valsalva are identical but differ from that of either PDA or of AS-AR by features demonstrated in Figure 368.

LESS COMMON VARIETIES OF CONGENITAL MALFORMATIONS

Ventricular extension into the abdominal wall, a tube-like myocardial cul de sac communicating with one or the other ventricle, has been described a few times. Parsons (1184) wrote: "A systolic thrill and a continuous murmur could be detected over a smoothly rounded, pulsatile swelling immediately deep to the umbilicus." Powell (1231A) found systolic and diastolic murmurs and thrills in a case of congenital diverticulum of the left ventricle in a 17 year old Bantu.

Chiari's network (remnants of the valves of the sinus venosus) occur in the right atrium (259, 594, 1599). It is thought that thrombi sometimes form in the meshes of these networks and result in pulmonary embolism. The only other aspect of clinical significance is the generation of a murmur which may be mistaken for some more grave lesion. Alvarez and Herrmann (18) state that in one of these cases "a most peculiar low pitched thonging hum was heard along the right sternal border from the third rib region downward. The hum was musical especially in diastole and faded off into a distant purr in systole." There were similarities to a venous hum except that it persisted with pressure on the jugular veins. Autopsy revealed that the net traversed the inferior vena caval inflow tract. The patient also had syphilitic aortic regurgitation with a conventional murmur. Wilson (1571) described a case in which he thought the murmur of Chiari net resembled the *bruit de Roger*. The patient was a 40 year old man with

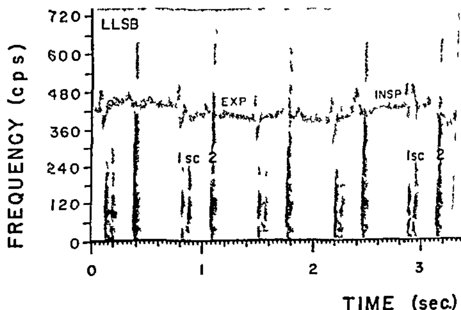


Fig. 422. Truncus arteriosus.
I. I. W. (B7470) 5 year old female shows at LLSB an early systolic click, unitary S_2 and absence of systolic murmur consistent with the diagnosis. The sonic findings are probably equally consistent with T/F with pulmonary atresia.

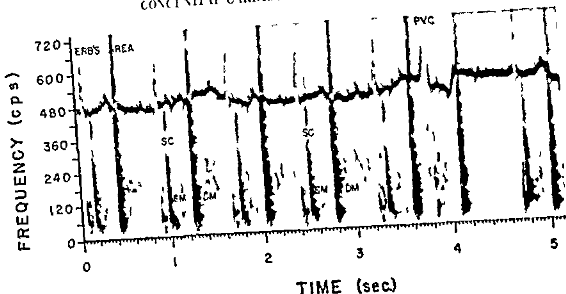


Fig 423 Truncus arteriosus

When I I (553836 A 48648) 11 year old male was 4 years old thoracotomy for presumed atypical PDA was performed and he was found to have a true truncus arteriosus The records show an early systolic click followed by a short systolic murmur which has a pattern consistent with an origin in high flow through the single truncus There is also a murmur of regurgitation at the arterial valve

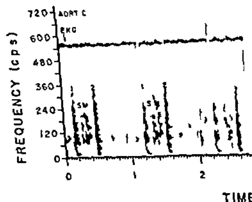


Fig 424 Anomaly of aortic arch

L B D (4 46 5) 22 year old female has a right sided aortic arch with retro esophageal course The systolic murmur was transmitted to the interscapular area The somewhat late onset of the systolic murmur and Christmas tree configuration are consistent with origin in the arch anomaly In childhood symptoms of aortic ring compression incorrectly interpreted as bronchial asthma had occurred

malignant hypertension as cause of death In the last week of life there was a continuous humming with systolic accentuation at the fourth and fifth left interspace Of course the location of the murmur was the only point of similarity to the *bruit de Roger* which is holosystolic not continuous It is of note that both patients had a separate variety of heart disease Although Chiari's net work is by no means a rare finding at autopsy (being placed at 1.5 per cent by Helwig (1169) and at 2 to 3 per cent by Yater (1598)) the production of an associated murmur probably depends on (1) a proper orientation of the network in relation to venous inflow to the atrium and (2) tensing of the network as a consequence of dilatation of the atrium

Truncus Arteriosus Communis

(Syn True truncus)

ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS In this malformation a single arterial trunk supplies both the pulmonary and systemic arterial trees One classification is based on the manner in which the pulmonary arteries leave this common trunk There is a single arterial orifice and a single arterial valve which according to some should have four cusps for the case to qualify as true truncus arteriosus There must of course be a large ventricular septal defect or a single ventricle

Truncus arteriosus is a variety of cyanotic congenital heart disease

CARDIOVASCULAR SOUNDS In some cases there are no murmurs The second sound is unitary as one would expect in view of the single valve However it may be prolonged and may be followed by

congested breasts—"the *mammary souffle*." Because of its accentuation in late systole, it appears to be arterial, not venous. It has a superficial quality and its superficial origin is further supported by the fact that it can be obliterated by pressure with the bell of the stethoscope. There is a fickleness about the murmur such that it appears and disappears more or less unaccountably. An intermediate grade of pressure with the stethoscope bell can intensify the murmur or cause one not previously present to appear. The murmur may have a musical quality. Knowledge of the mammary souffle (p. 233) and of the characteristics mentioned is sufficient to distinguish this phenomenon from PDA.

The syndrome of VSD with AR may simulate *PDA*; *patients have been mistakenly operated on* for presumed PDA (e.g., J. R. 726671, 26 year old white female). As suggested previously (p. 362) it is possible that the murmur of the VSD-AR syndrome and that of ruptured sinus of Valsalva are identical but differ from that of either PDA or of AS-AR by features demonstrated in Figure 368.

LESS COMMON VARIETIES OF CONGENITAL MALFORMATIONS

Ventricular extension into the abdominal wall, a tube-like myocardial cul de sac communicating with one or the other ventricle, has been described a few times. Parsons (1184) wrote "A *systolic* thrill and a continuous murmur could be detected over a smoothly rounded, pulsatile swelling immediately deep to the umbilicus." Powell (1231A) found systolic and diastolic murmurs and thrills in a case of congenital diverticulum of the left ventricle in a 17 year old Bantu.

Chiari's network (remnants of the valves of the sinus venosus) occur in the right atrium (259, 591, 1599). It is thought that thrombi sometimes form in the meshes of these networks and result in pulmonary embolism. The only other aspect of clinical significance is the generation of a murmur which may be mistaken for some more grave lesion. Alvarez and Herrmann (18) state that in *one of these cases* "*a most peculiar low pitched* thonging hum was heard along the right sternal border from the third rib region downward. The hum was musical especially in diastole and faded off into a distant purr in systole." There were similarities to a venous hum except that it persisted with pressure on the jugular veins. Autopsy revealed that the net traversed the inferior venacaval inflow tract. The patient also had syphilitic *aortic regurgitation with a conventional murmur*. Wilson (1571) described a case in which he thought the murmur of Chiari net resembled the *bruit de Roger*. The patient was a 40 year old man with

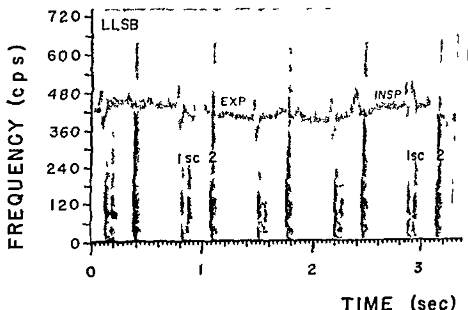


FIG. 422. Truncus arteriosus.

I. J. W. (B7470) 5 year old female shows at LLSB an early systolic click, unitary S_2 and absence of systolic murmur consistent with the diagnosis. The sonic findings are probably equally consistent with T/F with pulmonary atresia.

CHAPTER 17

Diseases of the Pericardium

ETIOLOGIC AND ANATOMIC CONSIDERATIONS Beyond the scope of this discussion is a detailed consideration of etiologic factors (1078) The anatomic patterns are of more pertinence to cardiovascular sound Acute fibrinous pericarditis has many causes but from the standpoint of sound generation there is little significance to anything except the exudation of fibrin on the opposing pericardial surfaces In the case of purulent pericarditis the fibrin in the exudate is probably responsible for the friction rub The friction rub in purulent pericarditis has no features distinguishing it from that of the fibrinous pericardites such as that of uremia or that of rheumatic fever

Among the chronic pericardial processes the two of pertinence aside from *chronic pericardial effusion* are pericardial adhesions and constricting pericardial scar so-called chronic constrictive pericarditis In the latter condition calcification occurs to a significant extent in roughly half the cases Usually the scar is generalized although it may be denser in some areas than others Fingers of scar tend to infiltrate among the myocardial bundles and the scar may be very dense in the AV grooves

PHYSIOLOGIC CONSIDERATIONS The pathologic physiology of chronic constrictive pericarditis has intrigued clinical physiologists for many years There is a variety of rather specific physical signs at least one of which is auscultatory and findings by means of special recording (see Fig 425) which can be considered collegative since all have a common basis in the main physiologic defect—impediment to diastolic filling of the ventricle (It is now evident that constriction of the ventricles is responsible for the clinical manifestation of chronic constrictive pericarditis that it is this portion of the heart which must be decorticated at surgery and that constriction of the atria and great veins is of limited significance) These colligative phenomena include the following

1 The protodiastolic sound (Lian's protodiastolic pericardial snap) which is produced by the abrupt halt in filling early in diastole

2 The flat top and W pattern of ventricular border movement as revealed by electrokymography or roentgenkymography The bases of this pattern as simplified one as compared with the normal (see Fig 425) are as follows

a The constricted heart is not free to make accessory rotational and configurational changes responsible for the squiggle during isometric contraction and isometric relaxation

b Ventricular filling is rapid because of high atrial pressure The filling limb is usually steeper than the emptying limb

c Ventricular filling comes to an abrupt halt relatively early in diastole and there is a standstill in ventricular filling during the remainder of diastole

3 The diastolic heart beat (1187) of Wood and colleagues (the *Spitzenstoss* of Skoda) refers to the pattern of the apex beat (or precordial impulse in general) which rather than displaying an outward movement with systole shows a retraction and in early diastole has a sharp outward movement This paradoxical apex beat has the same basis as the *FLAT RAY* pattern the diastolic heart beat represents a translation to the precordium of the abnormal pattern of ventricular motion A localized diastolic heart beat is identifiable in a minority of cases in my experience However in many cases it is possible to note by observing the sternum and left midprecordium obliquely a diffuse systolic retraction and a rapid

a short diastolic murmur. The last feature helps differentiate truncus communis from T/F which it may otherwise simulate (1108A). In some cases there is a loud systolic murmur resulting probably from high flow in the common trunk. In the syndrome of true truncus with large pulmonary flow (288), a continuous murmur identical to that of PDA may occur. There may be an early systolic click produced in the single arterial trunk. See Figure 423.

Vascular rings may sufficiently compress or distort the aorta to result in a systolic murmur heard over the base of the heart and the great vessels. See Figure 424 for such a case.

Transposition of the great vessels, including the Taussig-Bing complex (1461), is one of the four most frequent varieties of malformation found in infants. Because of an average prognosis for life of only months, transposition is rare in later childhood and unknown later. The auscultatory findings are not specific. They are the result of associated malformations (e.g. VSD, pulmonary stenosis) and of changes in the orientation of the great vessels to the heart and front of the chest.

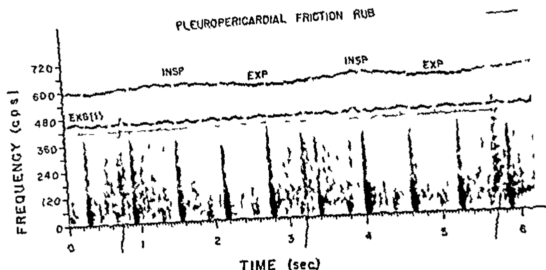


Fig. 436 Pleuropericardial friction rub

Aortic area in patient with advanced pulmonary tuberculosis. With inspiration a scratchy sound appears in the first part of systole. There is an inspiratory breath sound running over the heart sound in the region where the scratches are seen. At times pleural and pericardial rubs are difficult to distinguish because the beating of the heart may generate sounds in diseased pleura and pericardial friction may show pronounced variations with respiration. No evidence of pericarditis in this case.

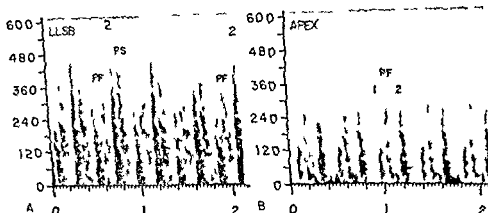


Fig. 437 Acute tuberculous *pericarditis* with effusion and moderate cardiac tamponade

C. C. (6 131?) ?? years old had tuberculous pericarditis with effusion. At the apex the friction rub is limited to systole. However it is easy to identify it as a rub rather than a *murmur because of its* quality as displayed here just as the ear can usually make the *same* identification and because of its circumscribed nature. At the *lower left* sternal border there is a loud circumscribed *sound in protodiastole which may* be a pericardial friction rub or possibly an early protodiastolic *sound of the type seen in con*strictive pericarditis but also seen in cases of pericardial effusion with tamponade (1078). As is usually the case this protodiastolic sound *occurs slightly* earlier than most protodiastolic gallops. However it *gives a gallop like* rhythm to the heart sound.

to the confusion which occurs between (1) *pericardial* effusion and (2) *myocardial* disease with flabby dilated ventricular wall

The heart sounds in *pericardial* effusion are usually muffled but may remain perplexingly loud in *some cases*. Mandani (1030) points out that if the intensity of the heart sounds when the patient is on his knees and elbows is compared with their intensity when he is on his back muffling of the heart *sounds* will be demonstrated

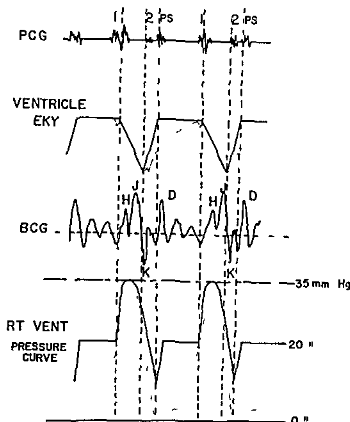


FIG 425 Chart correlating four early diastolic phenomena of constrictive pericarditis (1) early diastolic sound (ps) in the phonocardiogram (PCG) (2) flat top and V pattern of ventricular border movement as displayed by the electrokymogram (EKY) or roentgen kymogram (3) the abnormally large diastolic excursions (D) in the ballistocardiogram (BCG) (4) the early diastolic dip followed by diastolic plateau of the right ventricular pressure curve

outward movement in early diastole External pericardial adhesion without the constriction syndrome can produce a simulating pattern

4 The right[1] ventricular pressure curve obtained by cardiac catheterization shows (rather than the usual gradual rise of pressure in diastole through the range of only a few mm Hg) an early diastolic dip followed by a diastolic plateau early in diastole the pressure in the ventricle falls to a level approaching zero' but rises abruptly to end in a diastolic plateau at a high level pressure Obviously, this level is essentially *the same as that in the peripheral veins i e* as much as 25 mm Hg in many instances In fact, it is sufficiently *high that one may* speculate whether the pulmonary valve might reopen during late diastole because the pressure in the right ventricle exceeds that in the pulmonary artery This could probably happen only when the left ventricle is relatively free of constriction, otherwise diastolic pressure in the pulmonary artery is likely to be elevated

[1] That in the left ventricle has shown the same pattern whenever recorded

5 The water hammer" phenomenon when the *capacity of the ventricle is attained in diastolic* filling is responsible not only for the protodiastolic sound, the nadir or turning point of the early diastolic dip, and the end of the "V" of the 'flat top and V" pattern and of the outward movement of the diastolic heart beat, but it also produces abnormal early diastolic excursions in the ballistocardiograms In side to side ballistocardiograms these are most striking seemingly indicating that the force involved operates mainly in the lateral direction

Amyloidosis of the heart and diffuse myocardial fibrosis as well as endocardial sclerosis (so called "constrictive endocarditis (107a)) may so alter the compliance, and therefore the pressure *volume characteristics of the ventricle* that physiologic and clinical simulation of constrictive pericarditis results

CARDIOVASCULAR SOUND The *friction rub* of acute pericarditis has been fully described (see p 223) its transitory nature the necessity of listening in many areas in several positions of the patient on numerous occasions in cases of suspected pericarditis the triple character of the friction rub in full blown form but the isolated systolic or occasionally diastolic timing during early and late stages of its evolution the occasional musical quality like a wet finger rubbing on glass' the possibility of confusion for a to and fro murmur of aortic stenosis and regurgitation

Pericardial friction rub may occur with pulmonary emboli (112) It is the pleural surface that is rubbed by the beating heart There is usually, although not invariably respiratory variation in the pericardial friction rub of this type which in actuality is a pleuropericardial rub (Fig 426)

With pericardial effusion and tamponade there *may occur a protodiastolic sound* which probably has the same mechanism as that of constrictive *pericarditis (36) This sound endows the heart* with a gallop rhythm (Fig 427) and contributes

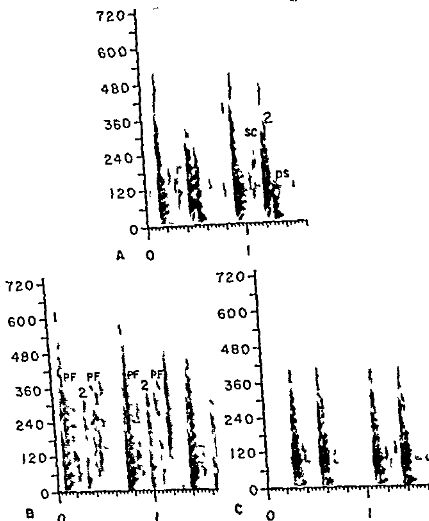


Fig 4?? Sounds in calcific constrictive pericarditis

A J P (6 3?f?) ag 1?? year Note the systolic click (SC) produced by the calcified plaques the slight splitting (S) in the second ... the early diastolic sound (PS) which situate ... to create a false impression of widely split S. B Pericardial friction rub early after pericardiectomy for constrictive pericarditis (I J ... C Later after operation at which time only slight rubbing sounds in systole and diastole persist

CONFUSING AUSCULTATORY SIGNS OF PERICARDIAL DISEASE Although well-documented in the world literature there is not general familiarity with several of the puzzling auscultatory phenomena of pericardial origin

Pericardial adhesions can produce systolic click and pericardial roughening can produce systolic murmur Not infrequently the two phenomena are associated the systolic click introducing the murmur The murmur of pericardial roughening is never holosystolic The murmur may be noisy or less frequently musical The systolic click of pericardial adhesion is usually mid or late systolic When in late systole it produces with the second sound a combination suggesting second sound plus opening snap of mitral stenosis—especially since such a late systolic click may develop after acute rheumatic fever with pancarditis The systolic murmur frequently occurs in the same clinical setting, i.e. after rheumatic fever and produces its own confusion—for the murmur of mitral regurgitation The musical late systolic murmur of pericardial roughening can be very loud and disturbing as a possible indicator of grave heart disease—which it is not (see p 207)

even with an effusion of small size. Normally, the intensity of the sounds is unchanged or increased in the knee-elbow position.

Colvin (280A) found, in cases of pericardial effusion, electrical alternans and auscultatory alternans without pulsus alternans. He suggested that effusion increases the mobility of the heart permitting pendular motion of the heart with a natural period of oscillation related in a 2:1 manner to the heart beat.

Usually a pericardial friction rub tends to disappear as pericardial effusion develops. However, the presence of a friction rub does not exclude the possibility of there being present a considerable amount of pericardial fluid.

When air as well as fluid is present in the pericardial sac, the so-called *bruit de moulin* or mill wheel sound results. This situation is rarely seen these days except on an iatrogenic basis, the air either being inadvertently introduced at pericardial paracentesis or being introduced intentionally for purposes of radiologic study of the heart and pericardium. The mill wheel sound was so named (*bruit de moulin*) by Bricheteau (174) in 1844. The Germans use the corresponding *Muhlengerausch* (712). In 1924 Stahl and Entzian (1431) described it when they removed fluid from the pericardial sac in a patient with tuberculous pericarditis and replaced it with air.

Bizarre sounds—often very loud—have been reported following perforation into the pericardial cavity by carcinoma of the esophagus (1144), by peptic ulcer of the esophagus (539) or stomach (619), by trauma occurring as an occupational hazard of sword swallowing (1182), or from foreign bodies, (such as mutton bone in the case reported by James (743)). Rupture of the esophagus into the pericardial sac, either through perforation of a peptic ulcer or penetration by a foreign body, produces a characteristic syndrome with severe substernal pain and shock and with a peculiar precordial murmur as an important feature. In 1849, Parkes (1182) recorded the melancholy case of a 19 year old sword swallower who some hours before death from this condition developed "a friction sound both diastolic and systolic . . . mixed with . . . a peculiar kind of metallic rhonchus carried up to the top of the sternum." Gellman and Silberstein (539) described the case of a 63 year old man with previous history of duodenal ulcer and sliding diaphragmatic hernia. He died 21 hours after onset of pain. There was a peculiar precordial murmur which could not be separated from the heart beat. One had the impression that liquid was rushing through a narrow opening. . . . Recovery with surgical intervention is possible in these cases if the diagnosis is made promptly.

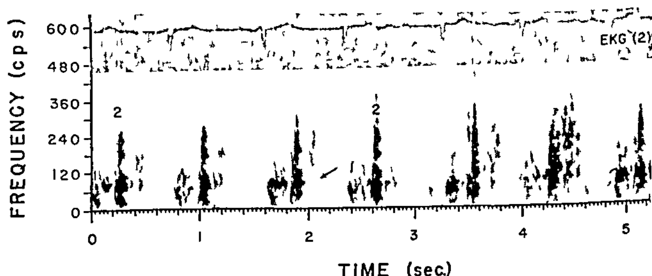


Fig. 428. Extracardiac early diastolic murmur or pulmonary regurgitation?

W. W. (494637) 82 year old male has no specific illness other than generalized arteriosclerosis and emphysema. There is a variable early diastolic murmur in the pulmonary area. Stethoscopically and spectrographically there is a suggestion of musicality. The characteristics of the murmur are compatible also with pulmonary regurgitation as described by Holldack and colleagues (699, 1125). Bourne described stethoscopically what seems to have been the same murmur and interpreted its origin as extracardiac.

CHRONIC CONSTRICTIVE PERICARDITIS The most characteristic single auscultatory finding is an early protodiastolic sound (930) It occurs closer to the second sound than a protodiastolic gallop (Fig 429) and is sometimes confused for a split second sound When the phenomenon called diastolic heart beat is present it is noted that the protodiastolic sound is temporally related to the end of the sharp outward movement of the precordium The higher the venous pressure, the more rapid is ventricular filling and the closer is the adventitious sound to the second sound In cases of early constriction there may in fact be variation in the S_2-extra sound interval this measurement being least at the end of inspiration The protodiastolic sound is *likely to be louder* and more clicking when there is extensive calcification in the pericardial scar It frequently exceeds the first and second heart sounds in intensity

The first heart sound is usually but not invariably diminished in constrictive pericarditis P_2 is frequently accentuated however, because a certain amount of pulmonary hypertension due to left sided constriction is frequent That all the heart sounds are not more regularly suppressed may be related to the improved coupling between the heart and thorax in these cases

Multiple clicks in systole are usually heard in cases with extensive calcification There may be a single click in the first part of systole about 0 11 sec after S_1 (490)

After pericardectomy several changes of note may occur in the auscultatory findings (1) A *mid-diastolic murmur* may develop (2) The protodiastolic sound is attenuated or disappears If *still* present it occurs later after S_2 Dunn and Dickerson (382) using the Q S_2 interval found a value of 0 43 before surgery and 0 55 after surgery (see Fig 84) (3) A split second sound frequently occurs and S_1 may also be split (4) Systolic and diastolic rubbing sounds suggesting systolic and diastolic murmur *may be heard*

In three patients two (E S and M C) teen age males and the *third* (*F G*) a female in her twenties I have observed after pericardectomy a third heart sound *followed by* a short rumble Functionally the patients are well However they have moderate cardiac enlargement Ven-

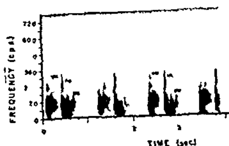


FIG 437 Carey Coombs murmur after pericardectomy for tuberculous constrictive pericarditis in E S (3 231") The heart remained dilated after operation The murmur had not been heard before operation

tricular dilatation without commensurate enlargement of the AV orifices is possibly the basis of the murmur I have not heard the murmur in question before operation This murmur can lead to a mistaken impression of associated mitral stenosis Probably rheumatic fever rarely perhaps never[*] causes constrictive pericarditis (318) (765) the incidence of a rheumatic valve lesion and constrictive pericarditis in combination can be expected to be the product of the incidence of the individual condition —a small value The possibility exists that some of the cases reported clinically as instances of associated mitral stenosis and constrictive pericarditis are *patients with* this particular type of diastolic murmur on the basis of constrictive pericarditis alone Lesine (885) points out that occasionally a faint prolonged third heart sound is *audible* at the apex resembling a faint murmur of mitral stenosis Jackson (740) described a *patient in whom* the murmur of mitral stenosis became evident after pericardectomy Whether *the patient* had true mitral stenosis is unknown Burwell (201) tells me of an autopsied case of the association of tuberculous constrictive pericarditis and rheumatic mitral stenosis *White and* coworkers (1541) described a series of cases with predominantly left sided constriction The *first* of the patients was said to show pressure on the mitral orifice by

[*] I (1069 10 8) have never encountered constrictive pericarditis in a case of indubitable rheumatic valvular heart disease Such *have however* been reported (318 765) these cases may represent coincidence however Calcific adhesive mediastinopericarditis without constriction is sometimes found in association with valvular heart disease

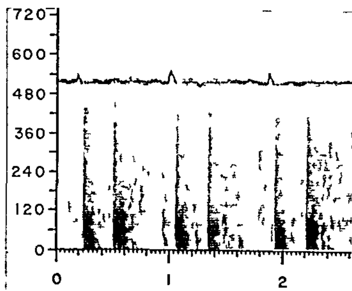


Fig 430 Large precordial thoracic cage defect

In this patient (J H 405305) the costal cartilages and a large portion of the body of the sternum were removed in the several stages of cardiac decortication for constrictive pericarditis The last operation was performed in 1951 and the patient is now free of symptoms of cardiac compression The heart lies immediately beneath the skin In most areas of the precordium sounds suggesting pericardial friction sounds or even endocardial murmurs are audible It is these which are displayed here The rub has three phases systolic protodiastolic and presystolic Residual roughening of the surface of the heart and its superficiality are considered to be the factors responsible for these sounds

There is essentially a syndrome (222 932 p 210) consisting of sticking pericardial pain palpitations and cardiac neurosis in association with pericardial clicks and murmur Traction of the heart on pericardial adhesions may be responsible both for the sticking pains and for the palpitation in terms of heart consciousness The neurosis is *often iatrogenic to a considerable* extent Because of ignorance of the true nature of the auscultatory findings the physician attaches more significance to the finding than it deserves Furthermore in the electrocardiogram residual T wave changes from previous pericarditis may be misconstrued as indicating coronary artery disease See Figure 431 Discovering characteristic systolic clicks assists identification of chest pain as pericardial, not coronary arterial, in origin

Diastolic musical murmurs of pericardial origin also occur I have encountered them after cardiotomy for mitral valvulotomy and without such intervention in patients with large hearts on the basis of rheumatic disease In these patients not only is there probably pericardial roughening but also the large heart rubs on its surrounding structures more than the heart of normal size

Occasionally I have encountered noisy murmurs in diastole which I thought because of their quality and timing and because of the absence of valvular heart disease were probably of extracardiac origin, specifically pericardial roughening For years following decortication for chronic constrictive pericarditis noisy diastolic murmurs on this basis may be audible (see later) and in individuals without cardiac surgery a similar murmur is occasionally heard Figure 428 presents the recording from the pulmonary area of an 81 year old man who had a diastolic murmur seemingly on this basis (Origin at the pulmonary valve cannot be excluded With normal pulmonary artery pressure the murmur of pulmonary regurgitation is thought to demonstrate this pattern at times Fenestration of the valve (p 273) is possible)

Melik Gulnasarian (1092) pointed out that the broad category of pericardial friction rubs includes both extra and intrapericardial varieties It is usually difficult to distinguish the two When extrapericardial in origin, the rub is likely to display more respiratory variation

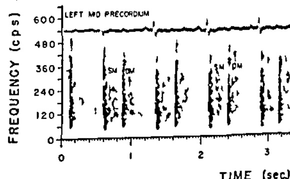


Fig 431 Simulation of endocardial murmurs for years after pericardectomy

Recorded at the area indicated not over the defect in M C (365313) in whom pericardectomy for constrictive pericarditis was performed nine years previously The patient has been functionally cured since operation Frequently after operation an impression of mitral stenosis was recorded because of an early or mid diastolic rumble at the apex At times even a presystolic murmur was described The patient has a large defect in the left anterior thoracic rib cage forceful pulsations of the heart are evident in this area

CHAPTER 18

Diseases of the Myocardium

ETIOLOGIC CONSIDERATIONS The etiologic factors in this group of disorders are too numerous and varied to discuss in detail. Ischemia deriving from coronary artery disease is the most frequent basis. Amyloidosis of the heart and diffuse myocardial fibrosis are pertinent to cardiovascular sound because the physical properties of the heart muscle are so altered that its behavior is strikingly similar to that of the heart in constrictive pericarditis.

CARDIOVASCULAR SOUND At least three characteristics of the sound in myocardial disease are noteworthy: (1) dull heart sounds, (2) gallop, (3) pulsus alternans with alternation in the intensity of the heart sounds and of murmurs.

Dull heart sounds are characteristic of myocardial disease but may be difficult to distinguish from the damped sounds of pericardial effusion. The dull second sound is explicable on the basis of low blood pressure. When the first heart sound is of poor quality, it is probably related to the relatively slow contraction of the ventricle with lower than normal development of intraventricular pressure. The faster the action creating a sound, the snappier is the sound. The converse is also true. Abnormally slow myocardial contraction in the preisometric contraction period is suggested by prolongation of the Q-1 interval in cases of myocarditis.

There is, unfortunately, a large element of subjectivity involved in judging of the quality of the heart sounds. A heavy muscular, obese or emphysematous thorax may also result in heart sounds of poor quality.

The first sound is often impressively attenuated in adolescents or children with myocardial involvement as part of progressive muscular dystrophy or Friedreich's ataxia. The finding is of particular note in the younger age groups. Kuhn and Holldack (822) described an attenuated first sound in 12 of 16 patients with myotonic dystrophy.

It is the protodiastolic gallop which is characteristic of myocardial disease, although with advancing heart failure a *presystolic gallop may also be present. Gallops are not easily heard* by the novice. Detection of faint gallops probably requires more experience than any other of the major auscultatory phenomena. Yet the clinical importance of the phenomenon as an indication of the state of the myocardium makes it very important that the clinician learn to recognize it. The auscultatory characteristics of gallops have been detailed earlier (see p. 174).

See Figure 433 *for description of the case of a* 27-year-old white male in whom extensive myocardial fibrosis from coronary artery disease was accompanied by atrial fibrillation, snapping M_1 and a mid-diastolic rumble apparently on the basis of dilated left ventricle. These auscultatory features led to the misdiagnosis of mitral stenosis.

Pulsus alternans occurs occasionally in myocardial disease. *It is best detected with the blood pressure cuff* and always should be quantitated in terms of mm Hg difference in systolic pressure between alternate beats. Pulsus alternans may be present only in the recumbent position. Probably the reduction in venous return in the upright position helps the competence of the heart—in effect, venesection or tourniqueting is performed. On the other hand, Friedman, Daily and Sheffield (484) found pulsus alternans in certain hypertensives only in the upright position. Or if the patient was recumbent, venous pooling with tourniquets or the Valsalva maneuver brought on pulsus alternans.

With pronounced pulsus alternans the heart sounds in alternate cardiac cycles may show al-

the calcified pericardium "resulting in definite mitral stenosis" In the 1890's Fisher (461) and Phear (1205) described a series of patients with a mitral diastolic murmur and neither organic mitral stenosis nor aortic regurgitation, many of the cases had what was then referred to as adhesive pericarditis

The confusion of constrictive pericarditis with rheumatic heart disease is always possible because in the former condition atrial fibrillation occurs in about one third of cases, cardiac enlargement, often marked, is present in over half (contrary to the "small, quiet heart" emphasized by earlier writers) the protodiastolic click may suggest a mitral opening snap, P is frequently accentuated, the left atrium may be quite large from irregular distribution of constriction, and, of course, the subjects may be young

The dilation of the ventricle present postoperatively in the patients with a diastolic murmur after operation is probably on the basis of disuse atrophy—the heart has in essence, been in a plaster cast for an appreciable period Acute dilatation of the heart after operation may occur is sometimes fatal and probably is often damaging Digitalization immediately after operation or even before operation is, in my opinion, indicated in such cases

After operation the early protodiastolic clicking sound may disappear or may be replaced by a third heart sound conventional in timing and quality The third heart sound may persist only during the period of cardiac dilatation in the first days or weeks after operation or may remain more or less permanently, as in the case illustrated in Figure 432, if the ventricular dilatation persists Many cases do not show a third sound at any time

In some cases, especially those operated on early in the evolution of the surgical technique for constrictive pericarditis and especially those cases which required more than one operation because of inadequate decortication the first time, there are systolic and diastolic friction sounds which can easily be confused for systolic and diastolic murmurs (see Figs 430 and 431) These are usually patients who have a large precordial defect in the bony thorax However, the "murmurs" in question are audible over intact portions of the chest as well as over the defect itself

ternation in intensity and there may be alternation in the intensity of murmur. In one study (484) 8 of 11 patients with pulsus alternans showed heart sound alternation as well. In seven cases of pulsus alternans another group (05) found concordant alternation of the first sound in all cases, concordant referring to louder sound related to stronger radial pulse. In four there was also alternation of the second sound, one concordant, three discordant. Jean and Racine (935) described alternation of aortic and mitral murmurs during left ventricular failure.

It is well recognized that with myocardial disorders regurgitation at the mitral or the tricuspid valve or both may develop.

In one patient (P. A. 314395), a 50 year old colored male with what eventually was proved at autopsy to be diffuse myocardial fibrosis, presumably a the result of myocarditis, a long, rough groaning, leathery systolic murmur at the left of the sternum in connection with the EKG changes displayed in Figure 434 led to the diagnosis of septal perforation following myocardial infarction. The murmur was accentuated by inspiration and there was a systolic pulsation of the liver. It is possible that the murmur was produced by relative tricuspid regurgitation, although an alternative possibility is pericardial origin. A murmur of pericardial origin would be likely to show similar respiratory variation. Autopsy revealed that the right ventricle was affected especially severely with almost paper thin state of its wall.

Myocardial infarction (1048, 1183) results in heart sounds of poor quality, diastolic gallops, a pericardial friction rub (pericarditis epistenocardica), a murmur of mitral regurgitation from dilation of the left ventricle or infarction of a papillary muscle with or without rupture of same, a systolic click or thud from systolic expansion of a ventricular aneurysm. During the acute stage a pericardial friction rub occurs frequently—in 20 per cent of the 50 cases in one series (1286), one third of cases in another (122), and as many as 85

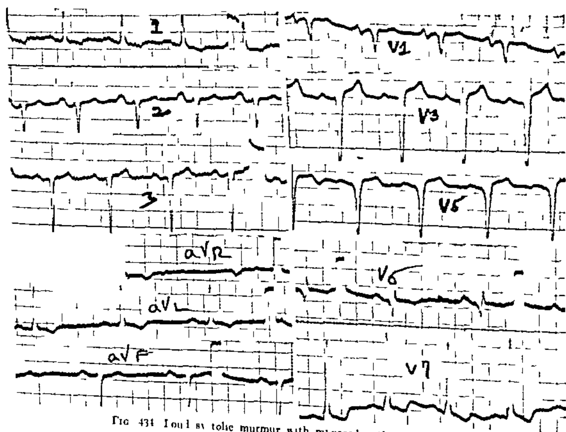


Fig. 434. Loud systolic murmur with myocardiopathy. (See text)



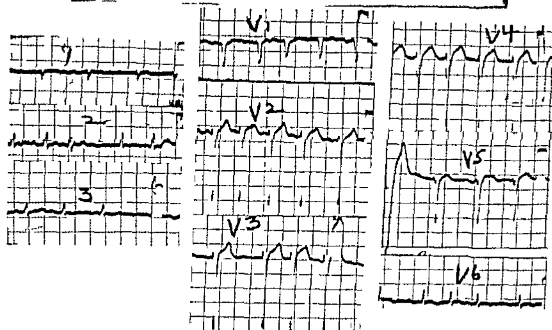


Fig 433 Diastolic rumble simulating mitral stenosis in patient with myocardial scarring from coronary artery disease

G B (752086) aged 27 years was diagnosed as having rheumatic heart disease with atrial fibrillation mitral stenosis and mitral regurgitation He was a strapping healthy looking white male weighing 228 pounds A faint apical diastolic thrill was described S_1 at the apex was described as snapping There were a grade I apical systolic murmur no opening snap and a late diastolic murmur which varied in length and intensity with cycle length Another observer described what he interpreted to be either a third heart sound or an opening snap The diastolic murmur was increased in the left lateral decubitus position Electrocardiogram showed bizarre pattern of the QRS complexes with low voltage and no R waves of appreciable size in any lead On left heart catheterization the left ventricle could not be entered from the left atrium Pressure in the left atrium was very high 40/28(?) mm Hg and the shape of the curves suggested mitral regurgitation At operation no stenosis was found At autopsy a few days later the mitral valve was perfectly normal the left ventricle was much dilated there was extensive coronary atherosclerosis and myocardial fibrosis secondary thereto

acute coronary occlusion. In one patient a soft, high pitched blowing presystolic murmur was heard in the fourth interspace just to the right of the sternum from the 10th to the 23rd day of illness. In a second patient a murmur of the same timing and quality was heard over the sternum and to either side at the level of the third, fourth and fifth interspaces. That this was an atrial friction would seem most likely. The authors thought, however, it arose through some interference with the tricuspid valve.

The complications or sequelae of myocardial infarction which are accompanied by auscultatory findings of note are pericarditis, ventricular aneurysm, rupture of the ventricular septum (1024) and rupture of a papillary muscle. The last accident is most likely to involve the left posterior papillary muscle and to occur with occlusion of the right coronary artery.

Perforation of the interventricular septum may occur within less than a day of the onset of symptoms (1338). In 1948 Fowler and Bailey (475) found 56 cases of ruptured ventricular septum in the literature and added two cases. Among the 40 of these 58 cases in which length of survival was known, 33 lived less than one month and 39 less than one year. Occasional patients live *longer* (533, 791): one remarkable patient *survived for* 58 months (1607). Of 46 cases examined 44 showed a systolic murmur which was usually maximal to the left of the lower sternum. In 24 it was accompanied by a thrill. Three showed a diastolic murmur as well. Large size of the defect was thought to account for the absence of murmur in one case (100) and profound shock in the second (73). (Nonpenetrating trauma to the chest is occasionally the cause of perforation of the septum (1420).)

The following features of rupture of a *papillary muscle* help differentiate it from rupture of the *interventricular septum* (1338): (1) the murmur is more bizarre, often diastolic and loudest at the apex; (2) a thrill is usually absent; (3) acute left ventricular failure is more likely to develop; (4) the murmur is absent surprisingly often. Akey (25) pointed out that a murmur is described in less than half the reported cases and emphasized the absence of thrill as a *rule*. He also was impressed in his own experience and that reported in the literature with the incidence of "pseudo-rub." He thought it in fact to be a murmur with an unusually superficial quality.

Sanders et al. (1311A) concluded that it is most often the posterior papillary muscle which is ruptured and that the location of the infarct is most often posterior. Septal rupture is more likely to occur with anterior infarction.

In a case of ventricular aneurysm following myocardial infarction we (1087) found an early systolic click. Mandini (1050) has had a similar experience. A midsystolic click has also been described (498, 882).

With ventricular aneurysm a strong precordial pulsation, sometimes localized to the region of the normal apex beat, sometimes more diffuse, with paradoxically weak *peripheral pulses* and distant heart sounds is characteristic. Systolic and diastolic murmurs heard in a localized area, usually at the site of the pulsation, have been described by many observers but are perhaps still too little known. Among those who have emphasized this finding are Scherf and Brooks (1356) and Paul (1186). Remlinger (1260) found a *musical quality to both* the systolic murmur which sounded like "ou" and the diastolic which sounded like "e". In five of 20 cases of ventricular aneurysm Wakil (1483) found a *fairly* loud and long murmur with a peculiar



FIG 436 Ventricular aneurysm which was associated with unusual murmurs (See text.)

with unusual murmurs. (See text.)

per cent in still other series (72) The rub occurs usually in the first week and most frequently in the second or third day It may be heard with posterior myocardial infarction as frequently as *with anterior infarction (1443)*, the pericarditis with any large infarction may be generalized An unusually prolonged pericardial friction rub should arouse suspicions of hemopericardium, a complication which occurs without anticoagulant therapy (20), but is more likely to occur when this therapy is used Organization of the exuded blood may be the basis for a systolic murmur, usually limited to late systole, after myocardial infarction Recurrent pericarditis with chest pain and friction rub, occurs in some patients after myocardial infarction without any repeat infarction

In 28 per cent of cases of acute myocardial infarction Shillito and others found a diastolic gallop (1386)

Castex (245) of Argentina described six cases of *anterolateral myocardial infarction* accompanied by a circumscribed systolic murmur in mid or late systole He suggested that the murmur is produced by the roughening of the endocardial surface—an untenable view in light of present analyses of the physics of cardiovascular sound It is likely that the murmur Castex described was of pericardial origin He quoted Huchard (see p 19) as considering a murmur of this type as a sign of cardiac aneurysm

Wolferth Wood and Margolies (1680) described an auriculo systolic murmur in the "tricuspid area" in two patients convalescing from

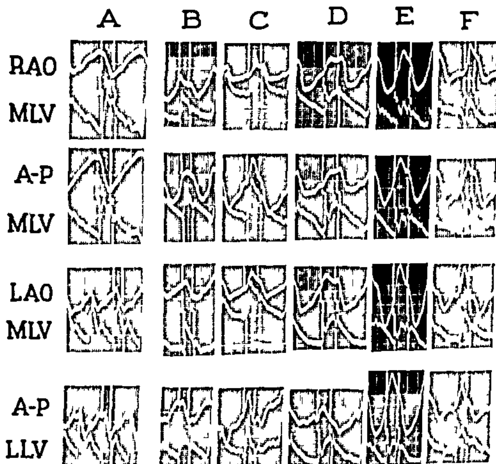


Fig 435 Electrokymograms (above in each recording) from the ventricular border in a normal subject (A) and *in four patients with myocardial infarction (B to F)* The carotid pulse (below in each recording) is used for timing purposes In the EKY a downward excursion indicates an inward movement of the heart border and an upward excursion an inward movement The point of importance is that the ventricular wall affected by an infarct tends *to show an outward movement* in systole whereas in early diastole it moved inward This is the familiar paradoxical motion of a ventricular aneurysm which may be present in milder form as a dynamic aneurysm as it were RAO right anterior oblique of the patient AP antero posterior LAO left anterior oblique MLV and LLV mid and *lower left* ventricular border respectively

CHAPTER 19

Systemic Arterial Hypertension

The second sound at the base is accentuated in systemic arterial hypertension. Usually A_2 is louder than P_2, but occasionally the opposite is true. Since the pulmonary second sound has contributions from the closure sound of both the aortic and the pulmonary valves, whereas the aortic sound is of unitary origin, it is not difficult to understand the seeming paradox of P_2 louder than A_2. The latter phenomenon is most frequent in young persons.

The second sound is not split in hypertension. Contrary to what seems to be generally thought, hypertension of either circuit, systemic or pulmonary, without bundle branch block or shunt, produces no impressive or consistent splitting of the second sound.

The mitral closure sound is usually delayed in systemic arterial hypertension. The Q-1 interval is prolonged although it is not as long as in most mitral stenosis. (An aortic ejection sound must not be confused for the mitral closure sound.) The mechanism of the prolonged Q-1 is not entirely clear. Possibilities are the following: (1) Through effects of hypertension on the myocardium, contraction may be slower during the preisometric contraction phase. In myocarditis the Q-1 interval is indeed likely to be prolonged. (2) Because of premature transmission of the atrial pressure wave to the ventricle, as suggested by the features of the presystolic gallop outlined on p. 179, the normal reinforcement of AV valve closure may be lacking in hypertension. A delay in mitral closure might occur for a reason similar to that for the delay in mitral closure in atrial fibrillation and with a prolonged PR interval.

The first sound may seem to be split because of the occurrence of an early systolic click and, of course, the presystolic gallop may, with S_1, produce a combination suggesting a split first sound.

Presystolic gallop is characteristic of hypertension (1016).[1] Although the connecting link was not then appreciated, it was the association of presystolic gallop with hypertension which led to its designation as "bruit de brightique" — sound of Bright's disease — by the French a century ago. See page 174 for a detailed description of the auscultatory characteristics of the presystolic gallop.

Occasionally the pulmonary hypertension resulting from chronic left ventricular failure in systemic hypertension is of such proportions as to result in a Graham Steell murmur. In one patient (W. A. 599694) in whom this was the case, the Graham Steell murmur showed accentuation with inspiration.

Relative aortic insufficiency from dilatation of the base of the aorta occurs rather frequently in arterial hypertension. It is difficult to estimate the frequency. Garvin (526) placed the incidence at 14 in 200 autopsied hypertensives—7 per cent. White (1539) cites the figure 4 per cent. Amyl nitrite may eliminate the aortic diastolic murmur of this causation, but it is doubtful that the effect is specific. Aortic regurgitation may occur if there is dilatation of the base of the aorta due to cystic medial necrosis secondary to the hypertension (or due to syphilis or the Marfan syndrome). Obviously it is also likely to occur if there is an associated congenital bicuspid aortic valve or a rheumatic lesion of the valve which of itself might produce little or no murmur.

[1] It is shocking to find the author of one monograph on hypertension equating presystolic gallop to split first sound (1366). The only justification is the auscultatory similarity. The recent demonstration of Leonard and colleagues (8 8) that a normal atrial contribution to the first sound moves back into presystole and constitute the presystolic gallop of hypertension (see p. 176) provides gratuitous justification.

'cooing,' 'plaintive' or musical character, occupying the whole of systole and early part of diastole, best heard at the site of the cardiac impulse, and not conducted in any particular direction" Three possible mechanisms for this musical murmur—as well as the noisy ones—exist (1) mitral regurgitation, (2) flow of blood in and out of the aneurysm, (3) pericardial friction

If the patient survives, the murmurs may diminish possibly because the aneurysm fills with clot Scherf and Brooks (1306) have emphasized a presystolic accentuation of the diastolic murmur Mitral stenosis has been mistakenly diagnosed in these cases *In the phonocardiograms* presented by Scherf and Brooks (1306) the systolic murmur was in the first part of systole and the diastolic murmur followed the second sound immediately These findings are consistent with the view that the systolic murmur is produced by the expulsion of blood from the main ventricular cavity into the aneurysm, and the diastolic murmur by the aneurysm bleeding into the ventricle," i e, emptying itself into the ventricular cavity when the pressure in the ventricle falls below that in the aneurysm (see Fig 435 for EKY demonstration of this pattern of ventricular border movement) This mechanism finds difficulty in accounting for the presystolic accentuation Actually, the phonocardiographic evidence for presystolic accentuation is not too convincing

Auscultatory changes with cardiac rupture have been described by Reznikoff (1267), Massey and Drake (1046, 1047), Nuzum (1148) and Bishop and Logue (106) In a patient in whom rupture occurred during auscultation, Massey and Drake (1046) stated that "no friction rub was heard before this spectacular episode but immediately after its inception an intense grating pericardial rub was audible from the third left *interspace down to the apex*"

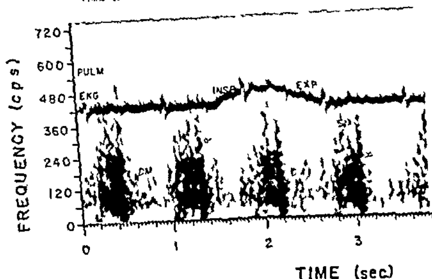
In a recent patient (G B, 763681) with anterolateral myocardial infarction it was noted that the apex beat had an unusually large outward excursion early in systole that in contrast the heart sounds were feeble and that a localized bulge was present at the lower left heart border on x ray The patient died from rupture of a ventricular aneurysm (Fig 436) 12 days after admission About 24 hours before death the apical pulsation was observed to be much more striking and a systolic murmur and early diastolic murmur of unusual quality were heard



Conceivably menstruation is accompanied by release of thromboplastic material in some women.

Any pulmonary hypertension which may be present is exaggerated by the presence of high flow in the pulmonary circuit—be it transient as with exercise or permanent as with septal defect.

The pulmonary valve seat is less substantiated



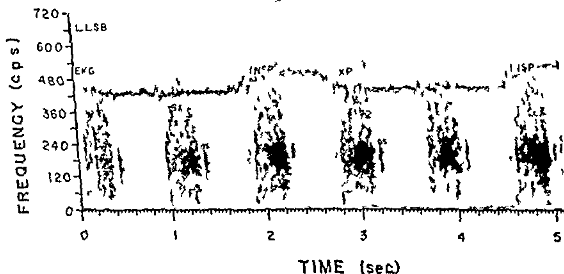


Fig. 438. Sound-like opening snap in primary pulmonary hypertension.

N. N. 16 …3 year old white female was thought clinically to have *primary* pulmonary hypertension. The clinical condition was confirmed at autopsy which revealed pulmonary arteriosclerosis and arteriolosclerosis. The patient had had two pregnancies. The symptoms were syncopal attacks, ascites, hepatomegaly and edema. Electrocardiographically confirmed. At *autopsy* there were evidence *of marked* right ventricular hypertrophy.

Heart sounds were recorded twice at 4 month intervals with identical results. There is a holosystolic murmur and an early *diastolic* snap. The murmur may be that of tricuspid *regurgitation*; there were prominent pulsations in the neck. The early diastolic click is of uncertain genesis. Evans (411) has described a similar finding. It is probably a tricuspid opening snap. Both the mitral and the tricuspid valves were normal at autopsy.

CHAPTER 20
Pulmonary Arterial Hypertension

The most common cause of pulmonary hypertension is increased back pressure from the left side of the heart from many different causes including systemic arterial hypertension—just as left sided heart failure is the most frequent cause aspect of cystic fibrosis of the pancreas, pulmonary fibrosis of many causes etc, etc) Primary pulmonary hypertension is legitimately considered a separate category

Primary pulmonary hypertension occurs with

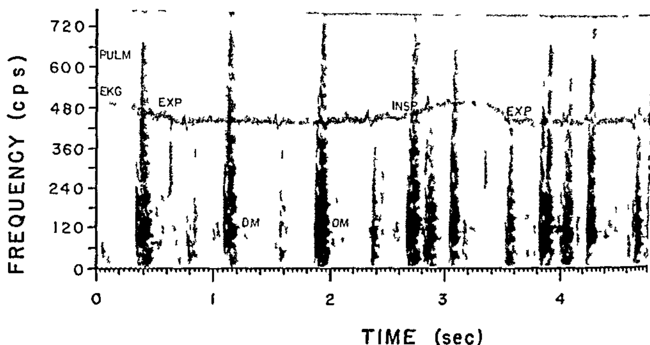
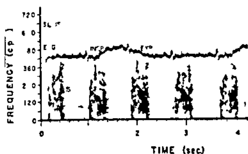


Fig 437 Multiple pulmonary emboli

Pulmonary area in I S (355061) 69 year old female with pulmonary hypertension from multiple emboli Atrial fibrillation S is greatly accentuated with slight splitting especially in inspiration the second (pulmonary) component is much the louder S_1 is almost absent except after a very short diastole There is a Graham Steell murmur

of right sided heart failure Multiple pulmonary embolization is an increasingly well recognized cause (1173) Other causes include (1) congenital malformation of the pulmonary vasculature (fetal persistence) usually in combination with septal defect (2) wear and tear from increased pulmonary flow in ASD VSD and PDA and (3) cor pulmonale due to a variety of types of primary lung disease (e g sarcoid, the pulmonary

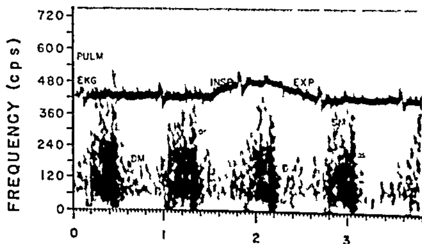
overwhelming predominance in females and serious symptoms develop usually at about the age of 30 years Rarely it occurs in children (1584) One of the more attractive suggestions is to the pathogenesis of many of these cases is that thromboplastic material is released into the blood stream and produces tiny fibrin clots which lodge in the lung The placenta especially if traumatized is a rich source of thromboplastic material



Conceivably merely menstruation is accompanied by release of thromboplastic material in some women

Any pulmonary hypertension which may be present is exaggerated by the presence of high flow in the pulmonary circuit—be it transient as with exercise or permanent as with septal defects

The pulmonary valve seat is less substantial



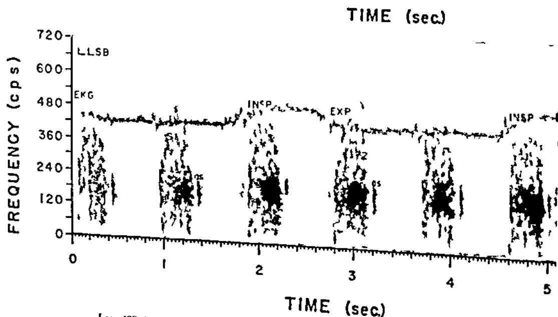


Fig 438 Sound like opening snap in primary pulmonary hypertension

V N (0 1551) 32 year old white female was thought clinically to have primary pulmonary hypertension The clinical conclusion was confirmed at autopsy which revealed pulmonary arteriosclerosis and arteriolosclerosis The patient had had two pregnancies The symptoms were syncopal attacks ascites hepatomegaly and edema Electrocardiographically confirmed by autopsy there were evidences of marked right ventricular hypertrophy

Heart sounds were recorded twice at 4 month intervals with identical results There is a holosystolic murmur and an early diastolic snap The murmur may be that of tricuspid regurgitation there were prominent pulsations in the neck The early diastolic click is of uncertain genesis Evans (444) has described a similar finding It is probably a tricuspid opening snap Both the mitral and the tricuspid valves were normal at autopsy

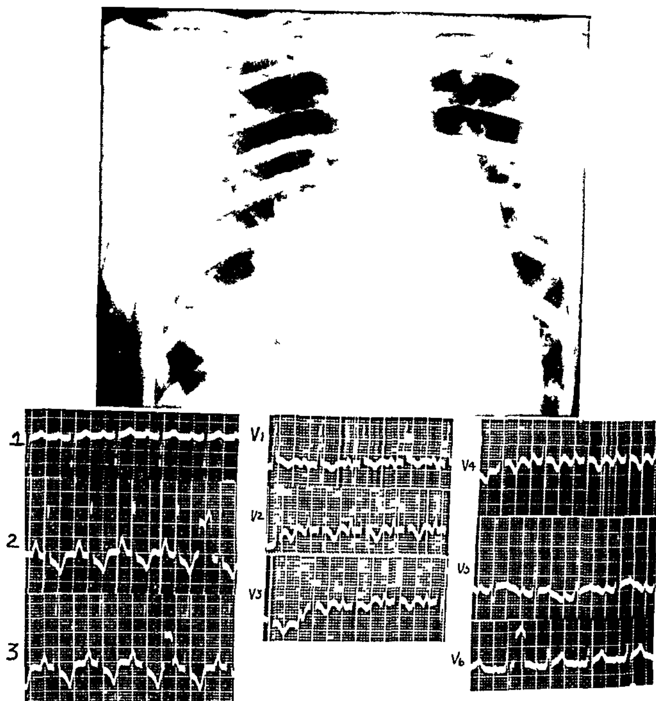


Fig. 439 Functional tricuspid stenosis with pulmonary hypertension (See text.)

than its aortic counterpart with the result that regurgitation with pulmonary hypertension is more likely to occur.

CARDIOVASCULAR SOUND. Many of the features are the same as for systemic arterial hypertension, e.g., presystolic gallop. The pulmonary second sound is accentuated *and many writers claim* that it is as a rule closely split. An early pulmonary systolic click is heard much more regularly than in aortic hypertension probably because the pulmonary artery is more superficially located. The early systolic click is usually followed by a systolic murmur generated in the dilated pulmonary artery.

The Graham Steell murmur (Fig. 437) was originally described as the murmur of high pressure *in the pulmonary artery* of whatever cause and not as an accompaniment of mitral stenosis alone. With severe systemic arterial hypertension of long standing I have observed (W. Y. 599691)

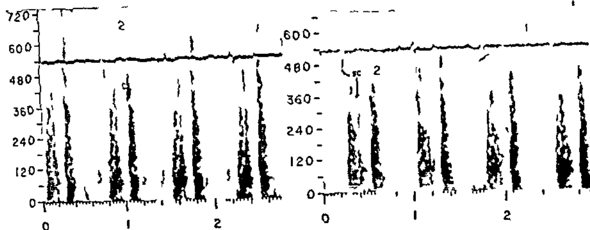


Fig 440 Primary pulmonary hypertension

(*Left*) Pulmonary area (*Right*) Apex S_2 greatly accentuated Early systolic click loud at ... S_2 split at apex (*right*) In this 6 year-old male (H K B14801) pulmonary arterial pressure was 97/73 mm Hg with a mean value of 85 mm The pulmonary artery was markedly dilated

Note (1) S_2 is accentuated in intensity and frequency ... but is not split (2) In the pulmonary area an early systolic click (SC) was understandably misinterpreted as a split S_1 It is mainly the tricuspid closure sound which is heard in this area (3) At the apex S_2 is truly split and the early systolic click is also demonstrated (4) An atrial gallop frequent in hypertension of either circuit is seen in the pulmonary area A faint third heart sound is seen in some cycle

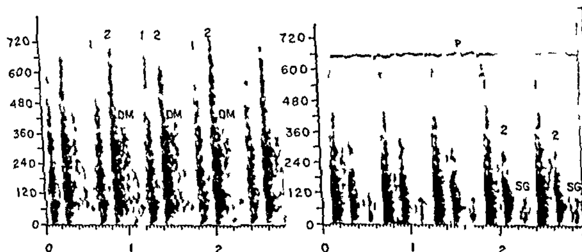


Fig 441 Primary pulmonary hypertension

(*Left*) Pulmonary area (*Right*) Apex Greatly intensified pulmonary closure sound with murmur of pulmonary regurgitation (*left*) Summation gallop at apex (*right*) the PR interval was 0.20 sec C H (68486) 29 year old female had a pulmonary artery pressure of 131/6 mm Hg with a mean of 89

what was interpreted as a Graham Steell murmur because it was closely confined to the left sternal border was accentuated by inspiration and followed a very loud P In general some additional cause such as multiple pulmonary emboli or pulmonary valve anomaly—both difficult to exclude—must be suspected in such case In one series (142a) of operated cases of mitral stenosis a Graham Steell murmur was found in 6 of 18 patients It disappeared in all following valvulotomy

Evans and colleagues (444) heard and recorded what they thought might represent an atrioventricular opening snap in one case of primary

pulmonary hypertension It was rather late after the second sound and may have represented an unusually snappy third sound See Figure 438 for a case of possible opening snap from our experience

In a 17 year old female patient in this hospital (B D, 745792 aut 26655) who had severe primary pulmonary hypertension, a mid diastolic rumble was heard at the apex and interpreted as representing the murmur of functional tricuspid stenosis heard in ASD Autopsy revealed no septal defect, (see Fig 439) Similar case of functional tricuspid stenosis in association with pulmonary hypertension have been described by a number of writers (see page 327)

CHAPTER 21
Miscellaneous Disorders

DYSRHYTHMIAS

The term *dysrhythmia* is used in preference to *arrhythmia* because some of the conditions discussed are not lacking in rhythm although they are disturbances of the cardiac rhythm, e.g. paroxysmal tachycardia.

SINUS TACHYCARDIA. Tachycardia of any type may be accompanied by accentuation of S_1. The ventricles are likely to find the AV valves in a wide-open position at the time of contraction. In addition, more rapid valve closure results in a snappier first sound with more components of higher frequency.

ATRIAL TACHYCARDIA. Although as a rule there are no particularly unique features of the heart sound, Figure 442 presents the finding of quadruple rhythm resulting from the presence of 2:1 block in a case of atrial tachycardia.

PREMATURE CONTRACTIONS (844). With any premature contraction, whatever the origin, the first heart sound may be delayed (61, 958, 1354) and accentuated because of wide open position of the atrioventricular cusps at the time of ventricular contraction. Schaefer and Little (1354) found that the first sound was more delayed relative to the QRS when it had its origin at the base than when it had its origin at the apex. Most often the PR interval is reduced in cases of atrial premature contractions. Therefore the AV cusps are likely to be widely spread at the time of ventricular contraction according to the considerations reviewed in connection with complete heart block (see p. 171). In the case of ventricular premature contraction, the AV leaflets may be widely spread merely because ventricular contraction occurs rather early in diastolic filling. In 20 cases Calo (217) found an accentuated S_1 in 6 cases, a diminished S_1 in 7, and an unaltered S_1 in 7.

In ventricular premature contraction both heart sounds are frequently split due to ventricular asynchrony (Fig. 445). The QRS is widened in ventricular premature contractions (PVC) just as in bundle branch block. A PVC arising in the left ventricle tends to have the pattern of right bundle branch block and vice versa. For some reason the heart sounds, especially the first, are more regularly split with PVC's than with bundle branch block. Splitting of the heart sound does not occur with atrial premature contractions because there is as a rule no widening of the QRS and no ventricular asynchrony.

In extrasystoles there is usually a direct relationship between the intensity of S_1 (Fig. 442) and the delay of that sound since both phenomena are based on the position of the AV valves at the time of ventricular contraction (302). However, *when* a ventricular extrasystole falls in the rapid filling phase of diastole, S_1 may not be accentuated despite a low position of the cusps due to poor filling of the ventricle.

There are several *reasons* (302) why the first sound is more often intensified and delayed in atrial than in ventricular extrasystoles: (1) Splitting of S_1 in VPC's divides or distributes the intensification. (2) Atrial extrasystoles usually have a short PR interval which is likely to result in a loud S_1. (3) Ventricular filling may be more complete in *atrial* extrasystoles because atrial contraction has contributed to ventricular inflow.

The QT interval (or the interval between the onset of ventricular systole and the first heart sound) varies with the PR interval (or the interval between atrial and ventricular systole). See Figure 446.

The second heart sound is usually reduced in amplitude or completely absent in extrasystoles (217).

pulmonary hypertension It was rather late after the second sound and may have represented an unusually snappy third sound See Figure 438 for a case of possible opening snap from our experience

In a 17 year old female patient in this hospital (B D, 745792, aut 2665a) who had severe primary pulmonary hypertension, a mid diastolic rumble was heard at the apex and interpreted as representing the murmur of functional tricuspid stenosis heard in ASD Autopsy revealed no septal defect, (see Fig 439) Similar cases of functional tricuspid stenosis in association with pulmonary hypertension have been described by a number of writers (see page 327)

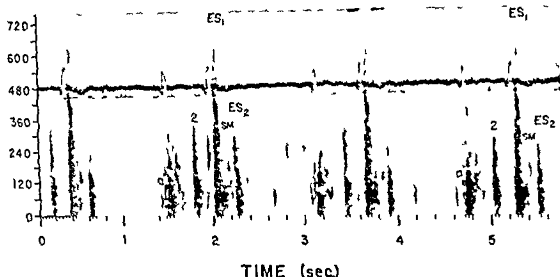


Fig 443 Bigeminy

Here is presented the recording from the aortic area in a patient (W C 15/817) with arteriosclerotic cardiovascular disease and pulsus bigeminus Note that the extrasystolic first sound is greatly accentuated—probably caused by the wide open position of the AV valves at the time the extrasystole occurs Note secondly that whereas in the normal cycle there is a sound which precedes the Q wave of the EKG, these are absent before the first sound of the ventricular extrasystoles The atrium is of course inactive in the case of ventricular extrasystoles unless retrograde conduction occurs In the third place note that the second sound is usually diminished in the extrasystole as compared with the normal sinus beat This is almost certainly related to low stroke output and low aortic (and pulmonary) diastolic pressure with the extrasystole Finally note the considerable abbreviation of systole in the case of the premature beat Systole is shortened mainly because of delay in the onset of the first sound This delay is probably on the same basis as that seen with atrial fibrillation and is related in some way to the absence of preceding atrial contraction The wider open position of the AV valves may demand a longer time for closure Note the 60 cycle electrical interference Also scattered through the record are four or five short sounds (snaps and crackles) which are easily identified as artifact

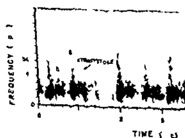


Fig 444 Effect of extrasystole on murmurs of AS and AI

In W W (10603) 28 year old male at the aortic area the Christmas tree *murmur* with the extrasystole is small but after the compensatory pause it is larger than normal The changes in the diastolic murmur are less definite

changing intensity of S_1 as a valuable diagnostic sign in atrial flutter Variability of S_1 is not always present but when it is discovered seems to have its basis in variable relationship of atrial to ventricular systole with variability in the position of the AV valves at the onset of ventricular systole In the electrocardiogram in flutter one observes fairly frequently beat-to-beat variation in the relation of the QRS to the saw tooth flutter waves Embry and colleagues (841) describe a variable triple rhythm in atrial flutter

In *atrial fibrillation* there is usually beat to beat variability in both the intensity of the first sound and its degree of lag after the QRS of the EKG (1113) This variability is secondary to the variability in the point in diastolic filling at which ventricular contraction occurs and corresponding variability in the position of the AV valves at that time (36 37 1247 1336) (see Figure 447) When mitral stenosis is combined with atrial fibrillation the variability in Q 1 interval is exaggerated (980) The high atrial pressure is responsible for delay in the first sound On the length of the preceding diastole depends how much the

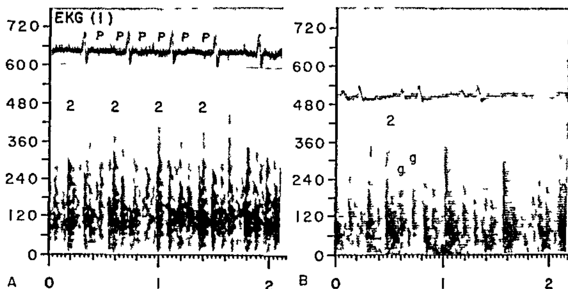


Fig 442 Quadruple rhythm
In the example presented in (*left*) (the aortic area) the 65 year old patient (C W 252981) had paroxysmal atrial *tachycardia with 2 1 atrioventricular block* Each atrial contraction was accompanied by a sound—one in systole one in diastole The second heart sound was slightly split in some instances The occurrence of an atrial sound in ventricular systole when the atrioventricular valves are certainly closed is evidence that tensing of the atrium and not movement of blood into the ventricle was primarily responsible for its production In the electrocardiogram shown here the presence of two negative P waves between each two QRS s is not clearly demonstrated but was clear from conventional electrocardiograms
In the recording at right (also from the aortic area and presented here by way of contrast) the quadruple rhythm results from the presence of both protodiastolic and presystolic gallop (g) sounds The patient has cor pulmonale caused by multiple pulmonary emboli The second sound is accentuated and is followed by a short diastolic murmur of presumed pulmonary origin P pulmonale is demonstrated by the electrocardiogram An early systolic click of *dilated pulmonary artery makes this a quintuple rhythm in some cycles*

A short early systolic murmur of low intensity may occur with ventricular premature contractions even though not present with normal beats The basis is not clear Atrioventricular regurgitation—which Little (956) shows does occur with extrasystoles before the AV valves are closed—cannot account for it since it is occurring after closure of the AV valves as indicated by the first sound Systolic murmurs of either the regurgitant or the ejection type are attenuated with the extrasystole (Figs 444 and 445)

The heart sounds in the cycle after the compensatory pause show differences from those in the average normal cycles The differences are the result mainly of the increase stroke volume which in turn results from the long diastolic filling phase The first sound is accentuated The second sound is also likely to be accentuated because of moderately increased pressure in both circuits Murmurs tend to be exaggerated in the post pause cycle (Fig 444) Alternation in the intensity of the heart sounds may parallel the pulsus alternans which may occur in the several beats following an extrasystole (217)

ATRIAL FLUTTER As a rule atrial sounds are audible only when complete atrioventricular dissociation is present With the combination of atrial flutter and third degree heart block, atrial sounds are almost invariably present as indicated by the relatively large number of reports (87, 101, 215 941 1317) Alternation in the amplitude of the atrial sounds related presumably to alternation in the strength of atrial contraction has been described (87) Hecht and Myers (660) found very loud atrial sounds in two cases of atrial flutter with AV dissociation The sounds were loudest in the pulmonic and aortic areas Interestingly they occurred during ventricular systole as well as diastole The authors raised the question of whether they might in fact represent atrial pleuropericardial clicks

Harvey and Levine (655) have emphasized

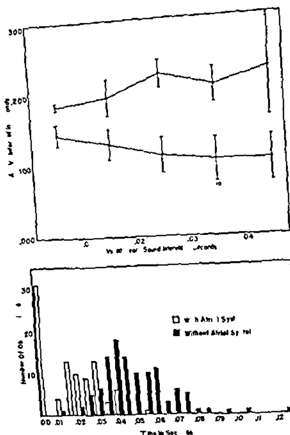


Fig 446 (Above) Relation between interval from onset of ventricular systole to the first sound (abscissa) and interval from atrial to ventricular systole (ordinate) Ventricular extrasystoles were induced by artificial means in dog (Below) Ventricular extrasystoles occurring without a preceding atrial contraction displayed a longer delay between the onset of ventricular systole and the first sound (abscissa) than did ventricular extrasystoles which occurred after an atrial systole (From Little Hilton and Schaefer (768))

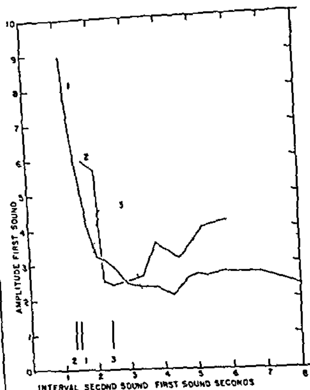


Fig 447 Variation in S_1 with S_2–S_1 interval in atrial fibrillation without mitral stenosis (Three cases) The lines indicate the timing of the protodiastolic gallop in each case (From Rytand (1137))

when S_1 occurred in early diastole rather than being accentuated as in subjects with atrial fibrillation and normal AV valve (Fig 448) Rytand proposed a diagnostic test based on this observation for use in cases of atrial fibrillation when the presence of mitral stenosis is uncertain Ravin and Bershof (1247) extended these observations They agreed that although a majority of cases of mitral stenosis (6 out of 10 in their study) and atrial fibrillation show no variation in the intensity of S_1 there are other patients (4 out of 10) who do show variation which is however of strikingly different pattern from that in cases of atrial fibrillation without mitral stenosis The intensity shows a slow decline with increasing length of preceding diastole reaching a minimum only at 0.5 to 0.7 sec values for the diastolic period The observers concluded that the patients with this phenomenon had pliant valves and low diastolic filling The question of the influence of cycle length on S_1 in cases of mitral stenosis and atrial fibrillation is complicated by the necessity to consider the mitral and tricuspid components separately (725A)

Medda and Scaravetti (1090) in 31 cases of atrial fibrillation in patients with various forms of heart disease (rheumatic mitral disease hypertension arteriosclerotic heart disease) could find no relation between the Q-1 interval and the duration of the preceding diastole (Fig 449) Intensity of S_1 showed a direct or inverse linear or biphasic relationship to the duration of the previous diastolic period—or no relation at all The clinical diagnosis was unimportant in determining which relationship was shown It did appear that in ranges of lower ventricular rates S_1 became

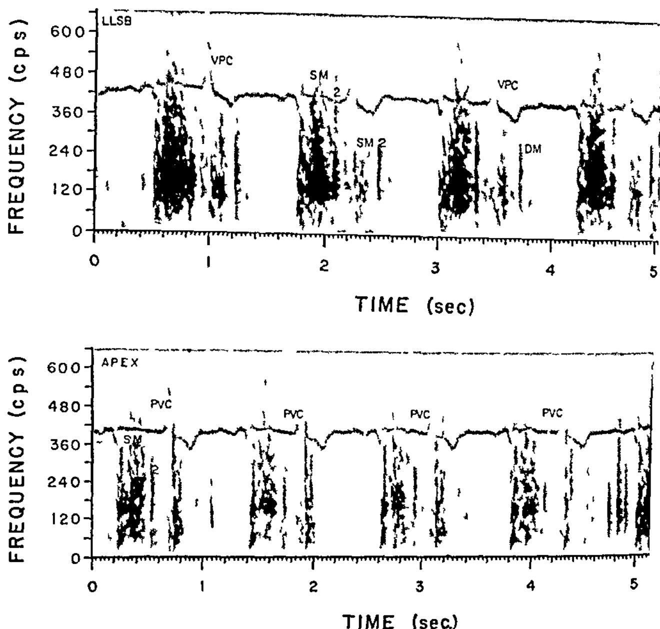


FIG 445 Bigeminy of digitalis intoxication

I A (562915) 54 years old had a history of syphilis treated eight years previously with persistent positivity of serologic test The systolic murmur at the base was transmitted into the neck and accompanied by thrill Fluoroscopy showed no dilatation of the aorta LLSB (A) and apex (*B*) The systolic murmur has the pattern typical of aortic stenosis which in this case was thought to have been only relative (p 264) S is followed by a decrescendo diastolic murmur which is interrupted by an extrasystole *Very little murmur accompanies the extrasystole In apex both* heart sounds especially the first are split with the extrasystole

atrium is decompressed by the time ventricular contraction occurs The longer diastole is the lower is the level to which atrial pressure falls and the less the delay of the first sound With shorter *preceding diastolic periods the first sound might* be expected to be louder (as well as more delayed) because the valve curtain is *bellied far toward the* ventricle Presumably because of a floating up of the valve leaflets as a result of ventricular filling a semiclosed position is attained and less noise produced during closure In patients with normal mitral valves minimal amplitude of S_1 occurred with diastolic periods between 0 20 and *0 25 sec (Fig 447) Thereafter there was at* times a secondary increase in amplitude However *Rytand (40) found that relatively little* variation in the intensity of S_1 occurred in patients with MS and that if anything intensity was less

In conclusion it should be pointed out that in addition to the particular changes in the heart sound noted above many of which can be detected by stethoscopy, auscultation is the most convenient bedside method for gauging the effect of carotid sinus compression in cases of tachycardia. A large portion of Levine and Harvey, *Clinical Auscultation of the Heart* (1949) was devoted to the auscultatory features of arrhythmia. To these authors is owed a general understanding of the distinctive behavior of each type of tachycardia (90) when pressure is applied to a carotid sinus area and then released.

Sinus tachycardia—gradual slowing and gradual return to original rate.

Paroxysmal atrial tachycardia—abrupt cessation of heart beat followed by sinus rhythm or abrupt resuming of previous rate.

Atrial flutter—slowing and return in jerky manner because of varying atrioventricular block.

Atrial fibrillation—slight effects at the most.

Ventricular tachycardia—no effect.

CONDUCTION DEFECTS

With *first degree atrioventricular block* manifested in the EKG by prolongation of the PR interval the first sound is likely to be muffled because of the relatively high position of the atrioventricular valve curtain at the time of ventricular contraction (p. 171). However with *the abnormally short PR interval of the Wolff-Parkinson-White syndrome* Levine and Harvey (888, p. 18) failed to find that the first sound is louder and snappier. On the other *hand in the syndrome of short PR interval with normal QRS complex and susceptibility to paroxysmal tachycardia*, Lown, Ganong and Levine (907) did note a snapping M_1.

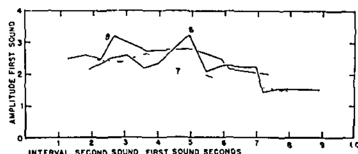
In *second degree heart block*, in which only part of the atrial impulses are conducted to the ventricle, there may be no particular auscultatory change. If there is Wenckebach *phenomenon*—progressive lengthening of the PR interval and final failure of conduction—the intensity of the first sound may show a progressive diminution. In 2:1 or 3:1 heart block with a slow ventricular rate (in the 40s) there may be an early diastolic murmur of the Carey Coombs type. This *murmur* may occur merely with very slow ventricular *rate*, but in the situation mentioned it is likely that the occurrence of atrial systole at the same time as rapid ventricular filling, or as passive basis, contributes to the murmur. If organic mitral stenosis is *present* with 2:1 block there may be two atriosystolic murmurs in diastole (278).

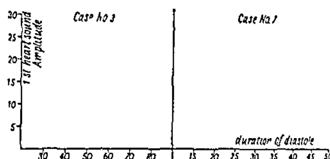
In *third degree heart block*—complete atrioventricular dissociation—there are cycle to cycle variation in the intensity of the first heart sound (76, 274, 378, 918), a systolic murmur and diastolic murmur(s) of note. Atrial heart sounds may also be heard and, as in most cases of AV dissociation of some degree, seem many times to be maximally audible at the aortic area and down the right sternal border in general. *Systolic echo* is an expression for the sound; the origin of the term is obvious.

When the PR interval is appropriately short the first sound may be very loud, the so-called *bruit de canon*. In young subjects there is likely to be intensification of S_1 with short PR interval (0.14-0.20 sec.) and with PR interval in excess of 0.32 sec. (Fig. 458). In older subjects the *second* zone of intensification may not occur (1331, 1582, 1578). Children (Fig. 454) resemble, therefore, the small animals such as the cat in which Dean (341) studied mitral valve motion (Fig. 455) and the dogs in which Boyer (see Fig. 129) studied S_1 intensity in surgically induced heart block. On the basis of a single case Vieira and Lilit (1489) claimed that when rheumatic mitral valve disease is combined with third degree block accentuation of the first sound may occur only with longer PR intervals of the order of 0.34 sec.

The variability in the first heart sound is related to the variability in the relationship of ventricular to atrial systole. Specifically there is every reason to believe that the variation in S_1 intensity is due to variation in the position of the AV valves at the *time of ventricular systole*. An older theory dates at least from 1912. Griffith of Manchester (597), an early student of the phenomenon, held that the accentuation of the first sound was caused by a coincidence with summation of atrial and *ventricular systole*. This theory would not explain the accentuation with longer PR interval and is on the whole untenable.

Laubry and Pudda (850) found that *bruit de canon* can occur with third degree heart block and a rapid ventricular rate. This is not surpri-





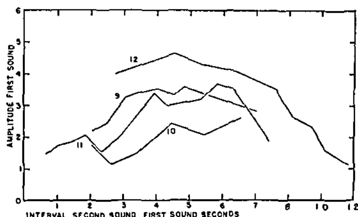


Fig 448 Amplitude of S_1 in atrial fibrillation with mitral stenosis

Note the relatively minor variability in the amplitude of S_1 If anything the intensity is less with shorter diastolic period (From Rytand (1336))

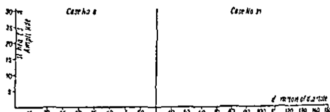


Fig 449 Relation of S_1 intensity to duration of preceding diastole

The two variables were significantly correlated in only 7 of 23 cases The direct relationship is illustrated above by case 3 an instance of decompensated mitral stenosis mean ventricular rate 73 per min The indirect relationship is illustrated above by case 7 an instance of myocardial infarction mean ventricular rate 10[illegible] per min No relationship exists in case 5 in which there was no other evidence of heart disease A biphasic relationship was found in case 21 an instance of combined mitral and aortic valvular disease (From Meda and Scanetti (1090))

louder when diastole was longer whereas at very rapid rates the converse relationship appeared to obtain

There is likely to be a short early systolic murmur of low intensity *This murmur may vary from cycle to cycle*

Variability is the rule in atrial fibrillation and the second sound diastolic murmurs and the mitral opening snap show it too The second sound may be *faint or absent if ventricular contraction* occurs so early in the preceding diastole that the arterial valves are lifted little or not at all Sometimes a paradox results The first sound is accentuated and the second sound is diminished or lost In tricuspid or *mitral stenosis the opening snap* and/or the onset of the diastolic murmur are closer to the second sound when the preceding diastole is short The intensity of the murmur of aortic or pulmonary regurgitation is greater in the cycle following a long diastolic *period* because of greater cardiac output and higher pressure at the onset of diastole

Some (1062) *have claimed an increased inci*dence of systolic murmur in association with atrial fibrillation as well as with ventricular extrasystoles and pointed to the observations of Daley McMillan and Gorlin (325) The absence of atrial *systole at the usual time just before ventricular* systole might explain regurgitation before closure of the atrioventricular valves but it is difficult to see how regurgitation — and murmur — could occur on this basis after the first sound

In ventricular tachycardia (886) *variability* in the intensity of the heart sounds is a characteristic feature Variability in the intensity of S_1 has its basis in a varying relationship of atrial to ventricular systole The presence of this variability *requires that the atria be beating independently* of the ventricles under control of the sinoatrial node or other supraventricular pacemaker Slight variability in rate is also a clue to the diagnosis The heart sounds are often so rapid and one *either the first or the second* so faint that there is risk of counting the rate at just half the correct one

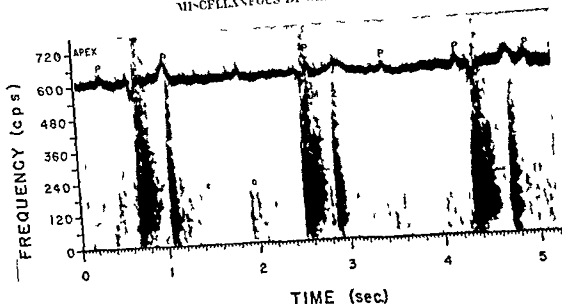


Fig. 452 Complete heart block
H J (51568) 60 year old man had Adam Stokes seizure There is an ejection type of systolic murmur probably caused by the large stroke volume Atrial sounds are faintly seen The one marked (a) seems to be double

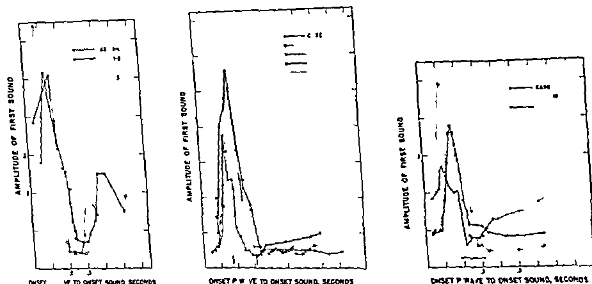


Fig 453 Variation of mean peak amplitude of S_1 against time of onset of S after P wave
(*Left*) Four children *Middle* Five adults in whom double atrial sounds were recorded the two vertical lines near the bottom represent mean timing of the two components from onset of P wave (*Right*) Four adults with atrial murmur at apex the horizontal line near the bottom represents the mean timing of the murmur from onset of P wave (From Rytand (1334))

cusps bring them in closer apposition with smaller excursion in production of the first heart sound

A *systolic murmur* has its basis in the large stroke volume which is necessarily present in heart block (Fig 452) The murmur has the characteristics of an ejection systolic murmur as outlined by Leatham (856) Specifically the murmur does not extend throughout systole but rather is separated from the second sound by a brief interval The *second sound* in complete heart block may be split if the idioventricular focus is situated in

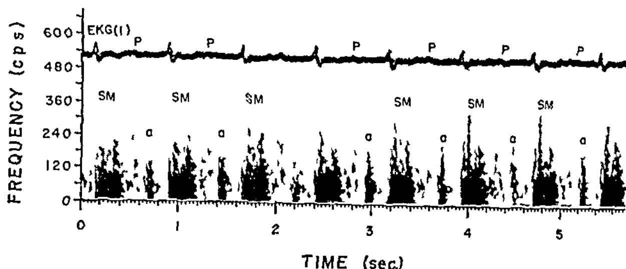


Fig 450 Prolonged PR interval in rheumatic heart disease

Apex in A. I. (299576) in whom the predominant lesions were aortic regurgitation and mitral regurgitation. The relatively faint early diastolic murmur is transmitted from the base. An atrial sound (or short murmur) is displaced into mid diastole by the long PR interval.

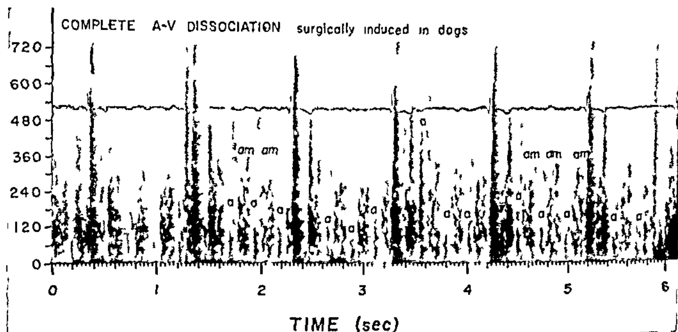


Fig 451 Surgically induced complete heart block in the dog

Third degree heart block was produced by Dr T. E. Starzl by the method he and his collaborators have described elsewhere. *A circumscribed sound (a) occurs with tensing of the atria and is followed by a murmur (am)* related presumably to the passage of blood into the ventricle. The occurrence of this phenomenon, an atrial sound followed by an atrial murmur, has been described in elderly patients with complete heart block (1335). Variation in the intensity and frequency span of the first sound is demonstrated. This record is too short to demonstrate convincingly that the louder sounds occur with the shorter PR intervals but such did appear to be the case. The recording was made one month after operation for creation of heart block. Although it seems unlikely, a pericardial origin of the murmur following each atrial sound cannot be unequivocally excluded.

ing inasmuch as variability of S_1 is seen with ventricular tachycardia, if there is a regular atrial beat (see p 438)

Stead and Kunkel (1435) observed that in cases in which it was possible to separate the effects of PR duration from that of the duration of diastole, *S_1 became fainter after longer diastolic periods.* Presumably floating up of the AV

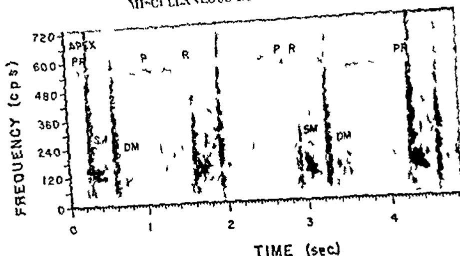


Fig 436 Congenital complete heart block
Apex in N V (B185 1) a year old white male who has complete atrioventricular dissociation as an isolated congenital anomaly Note (1) Variability in the intensity of S_1 depending on the preceding PR interval (2) the systolic murmur with the spectral characteristic of the Still murmur (3) the third heart sound which appears to be followed by a short rumble

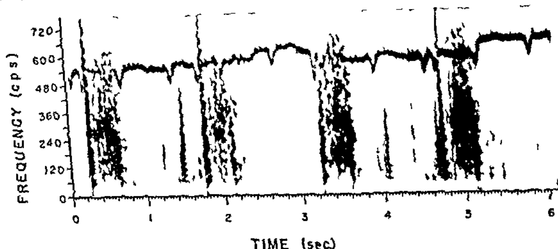


Fig 437 Pulmonary area in B C (44141) 14 year-old girl with pulmonary stenosis and congenital atrioventricular dissociation Systolic pressure in the right ventricle was 180 mm Hg An atrial septal defect was also demonstrated by cardiac catheterization Before operation an infundibular location of the pulmonary stenosis was considered likely because of the absence of post stenotic dilatation of the pulmonary artery On the day following the above recording resection of the infundibular pulmonary stenosis was successfully performed under hypothermia The recording demonstrates variability in the intensity of S_1 a systolic murmur which extends across the second sound (probably aortic closure sound) a double atrial heart sound An early diastolic murmur was present at the apex

and about 0 20 0 24 sec after the onset of the P wave If their interpretation that this wave is produced by momentary tricuspid closure is correct explanation for the atrial sound in these cases may be provided The Henderson Johnson mechanism—drawing in of the cusp in the wake of the ejection jet—may be involved in AV valve closure in such cases

Froment and colleagues (489) in writing on the atrial sounds in heart block (called "galop du bloc" by Louis Callivardin in 1914) point out the atrial sound may be single or double[1] depending on the position of atrial systole in ventricular

[1] Many workers (903) (31 S) have noted that the atrial sound is frequently double in complete heart block

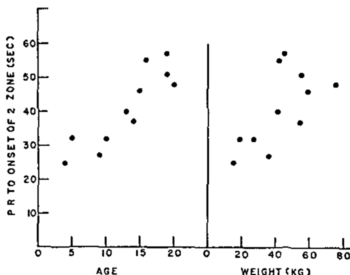


Fig 454 The relation hip between onset of secondary zone of S_1 accentuation and age and weight in complete heart block in man (From Shearn Tarr and Rytand (1382))

the ventricles at a site such that prolongation of the QRS results If the idioventricular focus is high '—close to the AV node—prolongation of the QRS does not occur and splitting of S is not to be expected With very slow ventricular rates an early diastolic murmur of the Carey Coombs type may occur on the basis of large AV valve flow and dilatation of the ventricle Wood (1590 p 324) states that it was present in over $^3{}_4$ of the present series [of congenital complete heart block], particularly when the rate was under 50 ' Paul and co workers (1187) found such a murmur in all six of the patients they studied and attributed it to high atrioventricular flow accelerated by recurring atrial systole '

A murmur in ventricular diastole following atrial contraction has been described by Rytand (1335) in elderly subjects In these cases there was often an atrial heart sound followed after a brief interval by a blubbering murmur the Rytand murmur Rytand referred to this as an atriodiastolic murmur The murmur had its onset after the onset of the P wave by an interval of 0 14 to 0 23 usually about 0 16 sec Windholz and Grayson (1573) in describing intrusion of the aortic root into the mitral orifice in hypertensive disease described three patients in whom this diagnosis was possible in life because of calcification of the fibrous skeleton of the heart All three had a diastolic murmur Two had heart block and were the same patients as those reported by Rytand It is possible that calcification of the annulus fibrosis mitralis and/or intrusion of the aortic root is responsible for the Rytand murmur However, Rytand's own explanation (1335) seems more likely "After auricular systole, normal mitral leaflets are floated nearly together In the aged, they remain longer and more fixed in this position because of their increased rigidity The murmur occurs then with continued forward flow through the relatively narrow orifice It is loudest in the part of early (ventricular) diastole which follows the phase of rapid filling ' Figure 451 shows both atrial heart sounds and the Rytand murmur in a dog with surgically induced complete heart block

Atrial heart sounds are heard in possibly 50 per cent of cases of complete heart block with the atrial pacemaker in the SA node Atrial heart sounds at a rapid rate are heard as a rule in case of complete heart block with atrial flutter a not infrequent syndrome In cases of heart block Fisch and Heeger (435) have pointed out a small sharp wave after the *a* wave in the jugular venous pulse

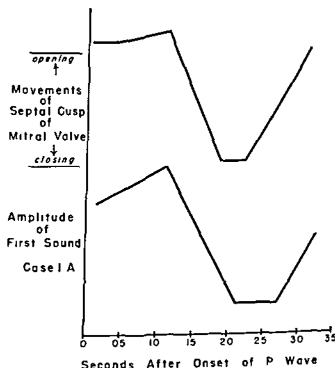


Fig 455 Curve (*below*) relating amplitude of first sound in complete heart block to time after onset of P wave (from Shearn Tarr and Rytand (1382)) and for comparison curve (*above*) of movement of anterior leaflet of mitral valve in perfused heart of cat (after Dean (341))

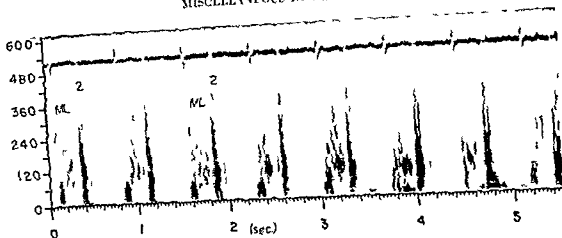


Fig 458 Mean Lerman scratch of thyrotoxicosis

This recording was made at the third left intercostal space of a patient with severe hyperthyroidism. A scratchy systolic murmur was present. In order to display better the scratchy quality of the systolic murmur the analyses were made with filter system D rather than the customarily used filter system C (p 60). The systolic murmur ends before the end of systole. Its mechanism is thought to be dilatation of the pulmonary artery with flow through it increased both in volume and velocity. This is an ejection systolic murmur which stops slightly before S2 in most cycles. The second sound is split in some cardiac cycles.

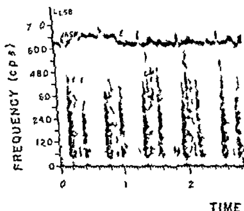


Fig 459 Lerman-Means scratch

LLSB in L D (376597) 46 years old with severe thyrotoxicosis. As in the case of pericardial friction rubs it is difficult to identify the physical basis of the scratchy quality. However as in pericardial friction rubs there is a rather diffuse frequency pattern and discrete transients can at times be identified. The murmur is not holosystolic.

of blood flow which contributes to the generation of the murmur is reflected by the reduced circulation time. Blumgart and colleagues (124) found that with an average increase in basal metabolic rate of 33 per cent the mean velocity of blood flow through the lungs was increased by 83 per cent. The increase in velocity of blood flow is out of proportion to that which accompanies a comparable increase in oxygen consumption from work in normal subjects (134). The *duration of ventricular systole* indicated in the phonocardiogram by the interval between the first and second *sound* is reduced more than would be predicted on the basis of tachycardia alone. Sensitization of the ventricle to adrenaline by thyroxin appears to be the basis. Wood (1300 p 880) states that a Carey Coombs murmur may occur in thyrotoxicosis on the basis of hypervolemic atrioventricular flow. On the other hand Likoff and Levine (917) state that they never heard a diastolic murmur in adult patients. One might expect to find it especially in children and adolescents with thyrotoxicosis since these age groups seem most prone to functional mitral stenosis. With this in mind I reviewed the cases of childhood thyrotoxicosis in the Johns Hopkins Hospital and found no case in which a diastolic murmur was described.

The *thyroid bruit* shows an accentuation in systole as a result of its basis in what is functionally an arteriovenous fistula. Venous hum in the neck is frequent in thyrotoxicosis but shows maximal intensity and frequency span in diastole after opening of the tricuspid valve. A continuous thyroid bruit occurs in about 20 per cent of

diastole In the patient they studied it was likely to be double when the EKG indicated a PR interval of 0.5-0.6 sec and likely to be single with longer or shorter intervals The second element was usually *loudest and was the one present when the gallop was single* Yet it was the first element which seemed to correspond to the usual presystolic gallop since the second element occurred about 0.20 sec after the beginning of the P wave rather than the 0.15 sec usual for the presystolic gallop

Stead and Kunkel (1435) and Wolferth and Margolies (1578) noted that the atrial sound (or auricular murmur, as they termed it) appears to vary with atrioventricular pressure difference Early in diastole this difference is maximal and the murmur loud, in the latter portion of diastole the difference is much less and the murmur either faint or absent The observation leads to the conclusion that the atrial sound in question is produced by (or in response to) the flow of blood into the ventricle and not by contraction of the atrium *per se* Bramwell (151) made similar observations on variable intensity of the atrial sound in a patient with complete heart block who was, however, thought by the author to have mitral stenosis, also

The auscultatory changes in *bundle branch block* are of note particularly in regard to the splitting of the second heart sound as outlined on pp 159 *to 167*

Braunwald and his colleagues (164, 165 168) found that, in 15 patients with right bundle branch block and evidences of right ventricular hypertrophy the onset of right ventricular contraction (as indicated by ventricular pressure curves) followed the onset of the QRS complex by normal intervals (0.045-0.075 sec) indicating that significant right ventricular conduction disturbance did not exist On the other hand in six subjects with right bundle branch block but without heart disease the electromechanical interval was prolonged (0.095-0.110 sec) confirming the presence of conduction disturbance In congenital bundle branch block splitting of the first heart sound is frequently found, whereas it is unusual in acquired forms of bundle branch block, facts consistent with Braunwald's findings

Contro and Luisada (286) have emphasized the rarity of splitting of S_1 in bundle branch block although splitting of S_2 is frequent They did find prolongation of the first sound in all of the 24 cases *they studied* Therefore they concluded that the first sound is normally too long a sound with much overlapping of its components, for one to expect separation of the components in bundle branch block

Earlier stethoscopic studies (794, 795) claimed a high incidence of splitting of S_1 in bundle branch block A presystolic gallop occurs rather frequently with bundle branch block, because of the underlying heart disease, and may create an illusion of splitting of the first sound (779)

With the Wolff Parkinson White syndrome splitting of S_2 is less consistently present than in bundle branch block (1374 1419)

THYROTOXICOSIS

The auscultatory findings in the heart in hyperthyroidism may contribute to simulation of other conditions, particularly mitral stenosis The first sound at the apex is often snappy which, with dilation of the pulmonary artery, straightening of the left heart border on x ray (Fig 461), enlargement of the left atrium atrial fibrillation accentuated P_2 and systolic murmur may lead to this mistaken diagnosis (947) (The PR interval of the electrocardiogram is sometimes prolonged) Quicker contraction of the ventricle is probably responsible (p 169) for the sharp first sound, the basis of this change is probably the interplay between thyroid hormone and adrenaline which is circulating or produced locally at the sympathetic nerve endings The scratchy systolic murmur at the left sternal border (Figs 458 to 460)—the Lerman Means scratch—can easily suggest pericarditis (1450) This murmur appears to be produced by high flow in the pulmonary artery dilatation of the pulmonary artery bring the site of murmur production to a superficial level accounting perhaps for its superficial quality (A different mechanism—in effect that it is indeed a rub—was proposed in 1920 by Goodall (567) who wrote as follows A superficial pericardial rub is often heard this is most common over the pulmonary base It is probably produced mechanically) The increased velocity

thyroid bruit is often so loud that a thrill is palpable or the murmur even audible with the stethoscope held a short distance from the skin surface. The murmur is usually rather invariable and is uninfluenced by stethoscope pressure, position of the head, or posture. The superficial location of the gland favors the appreciation of a thrill. Usually the bruit is well localized to the neck and even with substernal extension of the gland may have no significant radiation to the upper part of the sternum. *Kan and colleagues* (929) *claim the origin of the thyroid bruit is the superior thyroid artery* and that the area of maximal audibility of the murmur is at the upper pole of the lateral lobes. The site helps differentiate it from venous hum which is maximal in the area posterior to the sternocleidomastoid.

An interesting phenomenon is the persistence or even exaggeration of the thyroid bruit in patients treated with antithyroid drugs such as propylthiouracil and rendered euthyroid by objective tests (Fig. 462). These observations are consonant with the surgeon's finding of increased vascularity in the glands of such patients.

In thyrotoxic patients pistol shot sounds may be present over peripheral arteries as well as Duroziez and Traube signs.

ANEMIA

Chlorotic blood becomes a deceitful scene. With the blood of chlorotic patients as with the nerves of hysterical patients, it is necessary to guard against all sorts of frauds.

Duroziez 1891 (394)

There may be close simulation of mitral stenosis by the auscultatory findings in severe *chronic anemias* such as in sickle cell anemia (870, 1574) and hookworm disease (614). Mitral *snap* usually snapping and a striking diastolic *rumble* (416) may be present. A mitral opening *snap has* not been reported in anemia. However *it is* conceivable that such might occur since it has been described in other hypervolemic *conditions* such as atrial septal defect. If the differentiation of *organic* mitral stenosis *from* the heart of sickle cell anemia *is of critical* significance to the patient, right heart catheterization with measurement of pulmonary capillary pressure—or of course left heart catheterization with direct measurement of left atrial *pressure*—can be performed. The *first* sound is not delayed in anemia, another indication of whether organic stenosis of functional significance at least is or is not present.

Parenthetically it should be stated that so thoroughly has the medical profession been propagandized on the simulation of acute rheumatic fever by sickle cell crises and of mitral stenosis by chronic sickle cell anemia (805) that the shoe is now on the other foot. Rheumatic fever is going unrecognized and rheumatic valvular heart disease is not being so diagnosed because of the fact that the patient has the sickling phenomenon and anemia. At least six well established instances of the association of rheumatic heart disease with sickle cell anemia have been reported. Plichta and Speer (1213) cite four and add a fifth; L (1070) reported a sixth.

Venous hums are frequent in anemia; basis of the early *German* term *Nonnensausen* (nun's murmur). Carotid bruit in the neck is also likely to occur. Cephalic bruit may be striking especially if atherosclerosis is present. One writer refers to the case of a physician who had two massive gastrointestinal hemorrhages and with each had noises in the head.

A hemic murmur in *systole* in the pulmonary area is the most consistent single finding in anemia. It is caused by high flow in terms of both volume and rate in the outflow tract of the *right* ventricle and to reduced viscosity of the blood. The murmur may have a scratchy quality like the German Means scratch. But less frequent and more uncertain in its causation is a decrescendo blowing *diastolic* murmur beginning immediately with the second heart sound (Fig. 463). In 1907 Thayer and MacCallum (1471), after bleeding dogs repeatedly and *replacing* the blood with saline, heard what they *interpreted* as an aortic diastolic murmur. *In 1903* Richard C. Cabot (209) of Boston *in collaboration* with L. A. Locke described three *severely* anemic patients who had a diastolic *murmur* which was loudest at the fourth left costal cartilage and had all the auscultatory *features of that* of aortic regurgitation. The Cabot-Locke murmur must be uncommon but has been described in two patients by Porter and James (1222), in one out of 34 *patients* by Hunter (730), in one of 51 by Sanghvi *et al.* (1342) who found 17 instances of *mitral diastolic* murmur in the same group. Ellis and Faulkner (424) described a 35

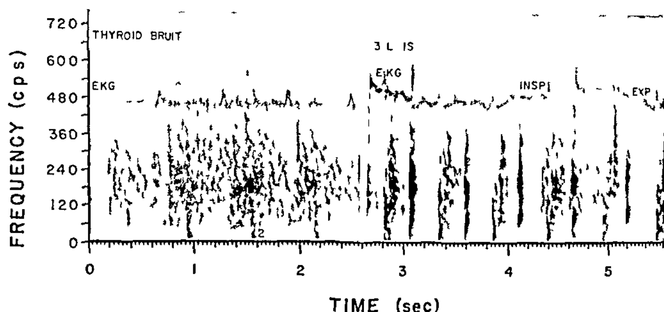


Fig 460 Thyroid bruit and Lerman Means scratch F W (69o137) 33 year old female had typical Graves disease



Fig 461 Dilated pulmonary artery in a 28 year old woman with *thyrotoxicosis* Fluoroscopically there was an increased amplitude of border pulsations (Courtesy of Cooley and Sloan (291))

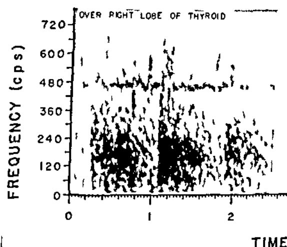


Fig 462 Persistence of thyroid bruit long after restoration of euthyroid state with thiourea compound J H (27139o) a female patient born in 1938 was first admitted in April 1956 with typical Graves disease A loud thyroid bruit was present Laboratory confirmation of the diagnosis included cholesterol 112 mg per cent BMR + 64 per cent butanol extractable iodine 18 2 µg per 100 cc serum She was treated with Tapazole One year later—April 1957—when the record here was made the patient was euthyroid The protein bound iodine had fallen to 4 0 µg per 100 cc serum Despite this the goiter and the bruit persisted The record shows this in striking form

cases a bruit limited to systole is present in the majority of the rest (929)

The nature of thyroid bruit as that of an AV fistula is well corroborated by the finding of a high degree of oxygen saturation (92 per cent) in the thyroid vein of thyrotoxic patients (1o82) At operation in past decades when patients were not so satisfactorily prepared for surgery as now the surgeon often found large veins which if cut squirted arterial blood rhythmically like arteries As with other murmurs of the arteriovenous type the thyroid bruit may be musical The

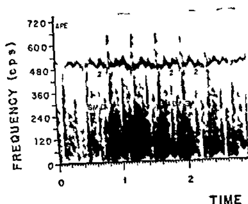


FIG 464 Carey Coombs murmur in anemia

Apex in J C (B34063) aged 20 months who suffered from severe nutritional (iron deficiency) anemia with hematocrit of 19 per cent hemoglobin of 2.3 gm per cent There is a decrescendo systolic murmur which stops slightly before the second sound Because of the rapid rate and short diastole the murmur is short However there is no question but that this is a murmur and not a gallop

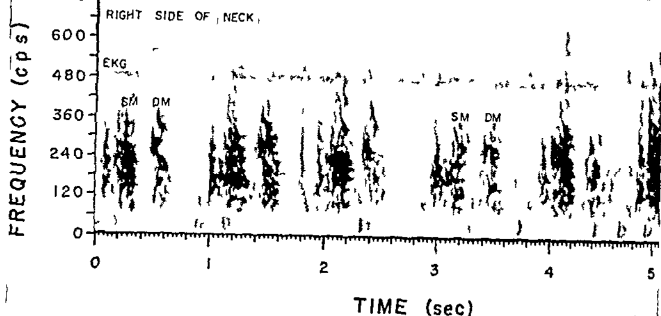
year-old woman with severe pernicious anemia hemoglobin only 8 per cent of normal and marked cardiac enlargment An aortic diastolic murmur disappeared with improvement in the anemia There are a few other similar reports (494 513 888 p 207 1574) Sometimes a venous hum transmitted over the upper part of the anterior chest may suggest such a murmur when the murmur is usually continuous A venous murmur which is largely limited to diastole does not begin with the second sound but after a brief interval at a point corresponding to the opening of the tricuspid valve Possibly at times the dilatation of the right ventricular outflow tract and pulmonary artery is sufficient to result in pulmonary regurgitation as was suggested by Gallavardin in 1908 (513) The presence in anemia of wide pulse pressure pistol shot sounds and capillary pulses increases the confusion with aortic regurgitation

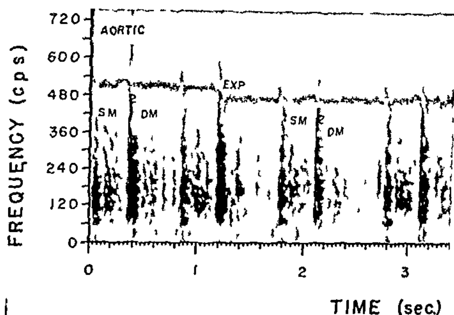
RHEUMATIC CARDITIS

In the pancarditis of acute rheumatic fever (ARF) auscultatory changes attributable to valvular myocardial and pericardial involvement occur as well as changes associated with conduction defects and arrhythmias

The most frequent valvular change is one producing an apical systolic murmur which may have a partially musical quality (Fig 465) Lunida (988) emphasizes the frequent presence of a harsher musical pulmonary systolic murmur which often has a diamond shape in the phonocardiogram What its pathogenesis may be and what its distinction if any is from the functional murmur described by Still (p 244) are uncertain Because of the high frequency of functional systolic murmurs in the same age group as that affected by rheumatic fever, Less of and Brigden (883) insisted that only a pansystolic murmur (or a diastolic murmur) can be taken as indication of valvulitis in acute rheumatic fever

The Carey Coombs murmur an early diastolic murmur usually following an accentuated third heart sound may be present in the early stages of ARF without permanent residua of mitral stenosis (30 97 113 115 1604) Wood (1590) states that a Carey Coombs murmur is present in 75 to 80 per cent of cases of rheumatic carditis It is not always easy to be certain stethoscopically or oscillographically whether one is dealing with a third heart sound or a short murmur The distinction can usually be made with the spectral PCG which usually will show in such cases either a circumscribed sound or a circumscribed sound followed by a short murmur The juxtaposition of third and fourth heart sounds may suggest a short murmur (1459) Friedman and Harris (485) found a diastolic murmur in 88 of 115 cases A murmur of aortic regurgitation does not develop as early as a rule and once it has appeared it rarely disappears completely Wood (1590 p 208) refers to its disappearance In one of my patients (P I 219593) evidences of profound aortic regurgitation were already present at the age of four or five years in the next four or five years signs of aortic stenosis progressed under observation as the aortic stenosis progressed the aortic regurgitation regressed to the point that no diastolic murmur was audible At death at 25 years there was necropsy evidence of trivalvular (aortic mitral tricuspid) stenosis The above sequence of events is an exception to the rule that once developed the murmur of aortic regurgitation persists Glazebrook (557A) described two patients in whom a murmur of aortic regurgitation appeared 20 days after the onset of acute rheumatic fever and dis





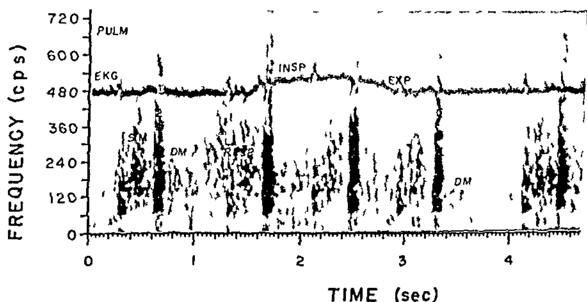


Fig 463 Severe and chronic anemia

Areas indicated in T C (B23255 aut 27582) 13 year old female with chronic evere anemia of the familial *Fanconi type (1251A)* In the neck (*top*) there are murmurs which may be partly of arterial partly of venous origin The component related temporally to ventricular systole is probably arterial in origin in the main whereas the diastolic one i venous In the aortic area (*center*) there are systolic and diastolic murmur In the pulmonary area (*bottom*) S is considerably accentuated and di plays normal *in* piratory splitting The tolic murmur has the pattern of an *ejection murmur (p 194)* There is a decre cendo early diastolic murmur The heart were normal at autop y

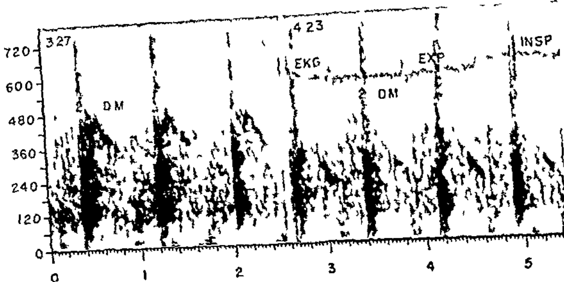


FIG 466 Musical diastolic murmur of SBE

Pulmonary area in J B (732861) 37 years old with SBE of the aortic valve caused by *Streptococcus viridans* on the basis of previous rheumatic affection The musicality superimposed on the conventional noisy murmur appeared midway in the course of therapy There is a single harmonic with a crescendo decrescendo pattern as in the murmur of retroverted cusp In the later record the harmonic occurs only in late diastole probably in relation to atrial contraction whereas in the earlier record the harmonic stopped abruptly at about the time of atrial systole A still later recording showed no musicality

heart endocarditis is less likely to cause murmur probably because of the lower pressures to which the valves are exposed For example the incidence of murmurless bacterial endocarditis of the pulmonary valve is higher than in the case of the aortic valve In a series of 23 cases of right sided bacterial endocarditis a significant murmur was absent in 15 (44%) In all 15 the correct diagnosis was not made

It is a trite rule not always remembered however that the association of murmur and unexplained fever of more than a few days duration spells bacterial endocarditis until otherwise proved In such cases in adults acute rheumatic fever presents one of the most difficult problems in differential diagnosis *(1313)*

The incidence of involvement of heart valves by SBE displays the same order as to relative frequency as for rheumatic valvulitis mitral aortic tricuspid pulmonic (634) Acute bacterial endocarditis may affect *previously normal* valves In the mitral valve rheumatism is the most frequent basis of change leading to SBE It is *mild* affection of the valve and of the heart which is most often complicated by SBE The usual story is that of a patient who either has known of no cardiac affection or has had an asymptomatic apical systolic murmur for many years The rarity of the complication of SBE in a patient with atrial fibrillation is another expression of the same fact In part the relative rarity of SBE in severe rheumatic heart disease is more apparent than real the severe cases are fortunately much less common than the mild cases severely affected patients die early and are not exposed to the hazards of intermittent bacteremia for such a long time Congenital bicuspid aortic valve is frequently the site of SBE The tricuspid valve is affected in mainliners morphine addicts who take narcotic by the intravenous route using crude nonsterile techniques The tricuspid valve may be involved *in rare cases* of anomaly of the valve The pulmonary valve is affected relatively frequently by the gonococcus

The murmur of regurgitation at any valve may have a musical *element (Fig 466)* when the regurgitation is on the basis of bacterial endocarditis Fenestration of a cusp or tear of a cusp probably results in a member which is free to vibrate musically Such a vibrant member appears to be a *sine qua non* for the production of musical murmurs in the cardiovascular system (I have a patient [J B 170645] with a musical systolic murmur of mitral regurgitation—fifteen years

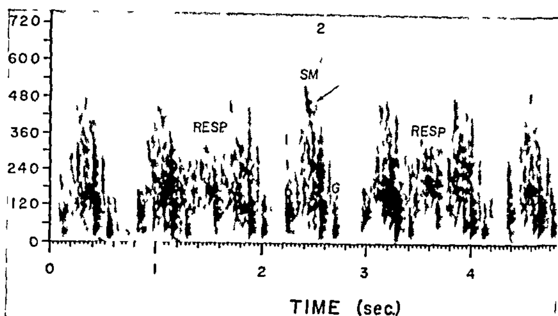


FIG 465 Musical crescendo systolic murmur in C C (681165) 14 year old patient with acute rheumatic fever Note the third heart gallop and the faint mitral opening snap between S_1 and S_2

appeared 2 months and 7 months later Feinstein and DiMassa (455A) comment on the rather frequent occurrence of an early diastolic murmur at the base in acute rheumatic fever and the subsequent disappearance of same Usually in their experience, such cases later show evidence of permanent cardiac damage and they emphasize the paramount prognostic significance of auscultatory findings

In 1951 Besterman (98) suggested the use of a vasopressor agent specifically phenylephrine to aid auscultation of the diastolic murmurs of early rheumatic valvulitis The elevation of diastolic pressure seemed responsible for the accentuation of aortic diastolic murmurs and probably reflex bradycardia with increased mitral flow accounted for the improved audibility of the mitral diastolic

The myocardial involvement of ARF is likely to result in dull heart sounds and gallops Whenever there is myocarditis, conduction defects most often prolongation of the PR interval may occur Many times it is possible to follow the clinical course of the patient by the intensity of the first sound which is inversely related to the PR duration Coombs (293) stated "In very acute carditis the first sound at the apex may become softened and almost inaudible", he ascribed the change to myocardial weakness Keith (775) was early to point out the more intimate relationship to PR duration

Pericardial friction rubs and residual systolic clicks are the usual expressions of pericardial involvement It is a well founded axiom that rheumatic pericarditis without endocardial murmurs is rare The late systolic click followed by the normal S_2 can suggest S followed by opening snap Familiarity with the late systolic click as a sequel of ARI usually is enough to make the differentiation easy It can be noted that it is the *first* of the two sounds which has the snapping dry quality It is too early in the clinical course for an opening snap to have developed The phonocardiogram assists in the differentiation The extra sound is too close to S_1 to be an S The true S_2 may be identified by the occurrence of normal inspiratory splitting The SPCG displays differences between valve closure sounds on the one hand and clicks or snaps on the other

Feinstein (455A) comments on the frequency with which diastolic murmurs are missed in acute rheumatic fever In the experience of his group when careful and frequent auscultation is practiced no other single sign is of as much prognostic significance as the auscultatory findings

BACTERIAL ENDOCARDITIS

A murmur due to regurgitation at the involved valve is the most frequent finding Cases of subacute bacterial endocarditis (SBE) without any murmur of any sort are reported (253 780) but must be very unusual On the right side of the

with production of a musical aortic diastolic murmur

Logue, Bahnson and I (1081) have observed a group of patients in which cystic medial necrosis of the ascending aorta with progressive dilatation and subsequent dissection occurred in association with calcific aortic stenosis of moderate severity. In all cases there was at least mild aortic regurgitation as well. We suggested that the valve lesion produced hemodynamic changes which resulted in cystic medial necrosis in the ascending aorta as a nonspecific morphologic expression of stress. It is a less likely possibility that cystic disease in the aorta and aortic valves was primary and was the *common denominator* in the calcific aortic valve disease and aortic aneurysm.

Noisy murmurs due to partial obstruction should be sought over the arteries particularly at points near their origin from the aorta. Double Korotkoff sounds over the right brachial artery but not over the left were noted by Calbraith and Norman (508). Murmurs should also be sought over the back. Evans and Curry (438) found a grade III systolic murmur along the dorsal spine maximal at about the level of the angles of the scapulae.

Pericardial friction rubs may occur from leakage of dissecting aneurysm into the pericardial sac. As demonstrated whenever air is introduced *into the* pericardial sac in cases of pericardial *effusion*, the pericardial reflection extends very high *on the* aorta, usually to *the take-off* of the innominate artery. *It is probable* that a fibrinous reaction with friction rub can be induced on the surface *of the aorta* when there is dissection in the media without there being *actual rupture* into the pericardial sac. Whether pericardial effusion of significant proportions can occur on this basis *is not* certain. In addition it is likely that actual leakage of *blood into* the pericardial cavity can occur and the *patient* survive for months or even year thereafter (592). The clinical picture of both of *the above* phenomena masquerades as benign idiopathic pericarditis. Pericardial friction rub is an important sign in both types of pericardial involvement.

Syphilitic aortitis is *rarely* accompanied by dissection but saccular aneurysm of course occurs commonly. A₂ is likely to be tambourine (216). This, like many of the other features of syphilitic aortitis such as calcification in the media of the ascending aorta extending to the sinuses of Valsalva and occlusion of arterial branches at the arch of the aorta and of the coronary ostia, is probably due to secondary intimal atherosclerosis to which the disease of the media renders the aorta exceedingly prone. The wooden quality of A₂ is probably due to fibrotic and atherosclerotic changes in the aortic cusps. Widening of the base of the aorta with *closure of* the aortic cusps through a greater than normal excursion may be a factor. It is *probable that the* base of the aorta can dilate to a certain extent without development of aortic regurgitation because of compensatory stretching of the aortic *cusps or adequate filling* of the orifice because of excessive size of the normal cusps.

EMBOLISM

Air embolism has been reported rather infrequently, mainly because its occurrence usually indicates an accident for which the physician may be responsible. From what clinical information is available and from observations in *experimental* venous air embolism in animals, a loud churning sound or mill wheel murmur is to be *expected*. It may be audible to the naked ear (391). It is important to recognize the pathognomonic *sign since* turning the patient into the left lateral decubitus may be *life saving* (391). Trapping of the air in *the right* atrium (1163) appears to be the mechanism of the beneficial effects which *have been* proved in human cases as well as in dogs. Aspiration of the heart has also been practiced (1432).

Hamby and Terry (626) in an article pointing out the risks of *air embolism* in neurosurgical operations performed *with the* patient in the sitting position described a *patient* in whom the mill wheel murmur disappeared promptly when he was turned *into the* recommended position and Musgrove and MacQuigg (1134) described a similar experience. Duboczky (375) described air embolism with mill wheel *murmur* audible to the unaided ear occurring during induction of pneumothorax. The sound ceased at once when the patient was *turned* in the left lateral decubitus position.

after SBE—caused by streptococcus viridans and cured with sulfadiazine Even the murmur of mild mitral stenosis has a musical element in this patient) The musical element is often changeable and may disappear a few weeks or months after it first develops (247, 1142) The abrupt appearance of a musical element suggests fenestration or tearing of a valve cusp Porter (1219) was listening to a patient as rupture of an aortic cusp occurred It is, of course, a cusp of the aortic valve which most often ruptures, however, aneurysm of the anterior (aortic) leaflet of the mitral valve with rupture and generation of a musical apical systolic murmur also occurs—witness the case of I F N (197522)

Although a musical element may be changeable, the murmurs in bacterial endocarditis are, on the whole less changeable than is usually taught "Changing murmurs" should not be insisted upon for the diagnosis

Lillehei found experimentally that bacterial endocarditis is likely to occur when bacteria are injected into the blood stream of animals with systemic arteriovenous fistulas (918) The heart valves are involved (Bacterial infection of arteriovenous fistulas may occur of course in man (633) and in animals) I have observed one patient (E McC 553929) in whom the Lillehei phenomenon appeared to be present there was a peripheral arteriovenous fistula and bacterial endocarditis developed on the mitral valve Curtin, Petersdorf and Bennett (321) described a similar case from the Johns Hopkins Hospital—a 26 year old housewife (F T 441128 aut 20932)

Tuberculous endocarditis has been reported

DISSECTING AND OTHER TYPES OF AORTIC ANEURYSMS AND SYPHILITIC AORTITIS

The varieties of musical murmurs which may occur in dissecting aneurysm have been outlined on page 220 Their abrupt appearance can be a valuable clue to the diagnosis Roberts (1284) reported a case which demonstrated in addition to the usual aortic diastolic murmur (see below), a systolic thrill of peculiar vibrating nature and a long, coarse, *whistling systolic murmur in the* aortic area and neck vessels At autopsy he found a prominent shelf like projection in the aorta just above the valve Noisy systolic *murmurs are commonly heard over the ascending aorta in case of* dissecting aneurysm

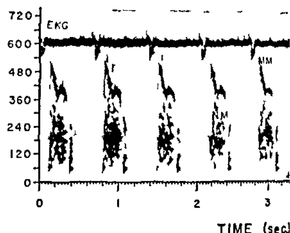
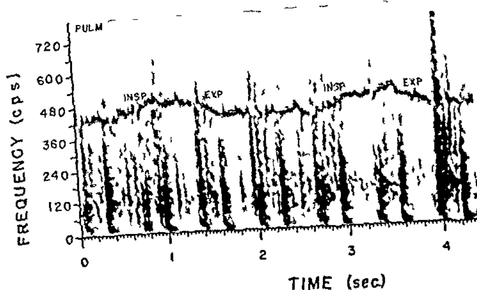


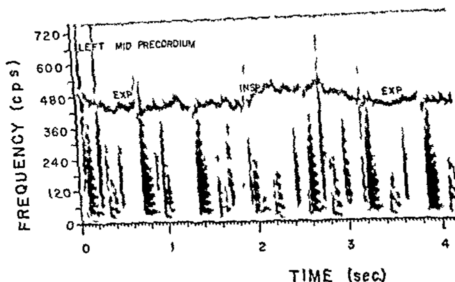
FIG 467 Dissecting aneurysm

Recorded in localized area at base of neck on right in A H (714091) with dissecting aneurysm and partial occlusion of the innominate artery The peculiar contour of the harmonic is puzzling

The appearance of an aortic diastolic murmur is *another useful diagnostic clue (1424) The sign* was first reported in the Anglo American literature in 1925 by Resnik and Keefer (1262) who *attributed the murmur to backflow in the false* channel In 1933 Louis Hamman (628) used this sign to make the clinical diagnosis of dissecting aneurysm—in a *CPC* to be sure—and suggested the now generally held explanation that distortion of the aortic ring by the medial hematoma results in aortic regurgitation Detachment of an aortic cusp (891 973) or lowering of an aortic cusp as a result of the intimal rent in the aorta above (1424) may at times be the mechanism *In many case* —possibly this is more often the case than the appearance of the murmur after dissection—the diastolic murmur antedates *the dissection and is* on the basis of idiopathic cystic medial necrosis the Marfan syndrome or hypertension Schnitker and *Bayer (1363) found that an aortic diastolic* murmur had been described in 24 per cent of reported cases In some cases e g, two reported by *Levinson et al (891) the murmur had a musical* quality This is not surprising since it is well known that the aortic diastolic murmur in *patients with the Marfan syndrome* may be musical furthermore cystic medial necrosis of the aorta without dissection may lead to partial detach*ment or fenestration* (658, 1051) of an aortic cusp



A



B

Fig. 468 Mediastinal emphysema

T B (39018) 14 year old boy has had bronchial asthma from the age of 2 years After an upper respiratory infection with cough the patient suddenly developed tearing chest pain There was subcutaneous emphysema in the neck Characteristic crunching sounds were heard The clicks responsible for the crunch (X) are unusually musical and are present in diastole as well as systole

During World War I it was noted (1413) that in penetrating wounds of the chest in the region of the heart there might be heard a sound of the nature of a click which varies from a faint sound heard by careful auscultation to a noise which may be compared to that heard in the earpiece of a telephone when the lever is moved up and down It may be heard sometimes when standing at the foot of the patient's bed Sometimes the patient is conscious of it Smith (1413) seems to have invented the expression pericardial knock

In 1928 Latter (934) described a 21 year old man who developed *spontaneous pneumothorax*

PULMONARY EMBOLISM

In pulmonary embolism a systolic murmur may suddenly appear over the pulmonary artery. The mechanism is probably most often acute dilatation of the pulmonary artery and infundibulum. On rare occasion it is possible that the embolus itself, lodged in the pulmonary artery or even the valve area, is responsible. Bunn (191) described a 55 year old woman who suffered multiple pulmonary embolization after the sclerosing of varicose veins of the legs. While he was listening, "there was the onset of a most unusual cardiac murmur. It was a harsh, grinding, continuous sound which varied in intensity but continued through systole and diastole. The intensity of the sound was greatly accentuated with systole. It had somewhat the character of an arteriovenous aneurysm in that it was continuous and rather roaring." The murmur had disappeared a few hours later at which time the patient had shock, hemoptysis and signs of pulmonary consolidation. She died 48 hours after the murmur was heard. Warburg (1508) had a possibly comparable experience. In a man with long standing rheumatic heart disease and atrial fibrillation whom he had many times examined, Warburg described the following episode. "During the last 24 hours he had heard a sound from his chest which he described as though something were dripping; he said that he thought his heart had burst. His wife was able to hear the sound when she was lying in the bed beside him. I was able to verify his statements. At every heart beat a clicking or slightly sonorous sound was audible in the room. A phonocardiogram showed that besides the sounds usually found in this patient there was a new murmur partly during systole, partly immediately after systole. . . . Four days later he had a typical pulmonary infarction with hemoptysis, stitch and fever. As soon as the infarction occurred he was no longer able to hear the sounds, and when I examined him the same afternoon the very loud new sounds had disappeared. He recovered in about ten days and since then I have had the opportunity of examining him numerous times but I have not heard the clicking and sonorous sounds. Most probably he had a loose thrombus in the right ventricle which later passed into the pulmonary artery."

McGinn and White (1065) wrote as follows in describing a series of cases of acute massive pulmonary embolism: "In two cases pericardial friction rubs were loudest in the second left interspace and are interpreted by us as probably due to the dilated pulmonary artery or distended right ventricle rubbing against the pericardium or with the pericardium pressing against the anterior chest wall."

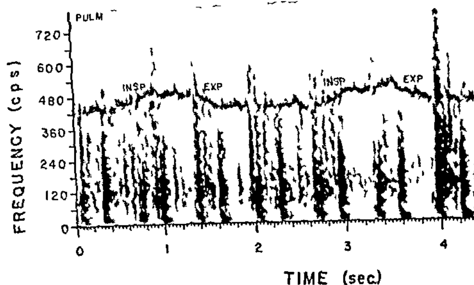
PNEUMOTHORAX, DIAPHRAGMATIC HERNIA, MEDIASTINAL EMPHYSEMA, PECTUS EXCAVATUM AND OTHER CHANGES IN STRUCTURES NEIGHBORING ON THE HEART

(including a discussion of some of the cardiovascular sounds which may be audible to the naked ear at a distance from the chest)

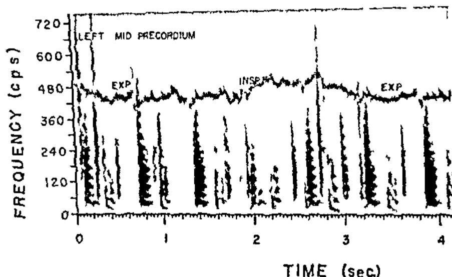
Systolic murmurs occur commonly with *pectus excavatum*. As to mechanism, distortion of the right ventricular outflow tract, pressure on a lappet of lung, and rubbing of pericardial surfaces, normal or abnormal, are all possibilities. Circumscribed systolic timing or extension of the murmur across the second heart sound are features suggesting a pericardial or cardiopulmonary mechanism.

Diastolic murmurs with pectus excavatum must be viewed with more suspicion. Conceivably distortion of the pulmonary valve area might be responsible. Since pectus excavatum of severe proportions can occur as one feature of the Marfan syndrome (1071), other stigmas of this disorder should be sought, particularly when a diastolic murmur is present. I have information on two patients (840) without definite evidence of the Marfan syndrome in whom a diastolic murmur of arterial type persisted after surgical repair of the sternal deformity.

In his delightful essay on "precordial noises heard at a distance from the chest" (74, 75) William Bennett Bean reviews many of the sounds produced by the action of the heart on surrounding structures which are in one way or another altered. These conditions include pneumothorax (traumatic, therapeutic, spontaneous), interstitial emphysema of the lung and mediastinum (spontaneous or traumatic), ectopic borborygmi (as with diaphragmatic hernia or with achalasia of the esophagus), hydropneumopericardium (see p. 416).



A



B

Fig. 165. Mediastinal emphysema.

T. R. (19918) 33-year-old boy has had bronchial asthma from the age of 2 years. After an upper respiratory infection with cough the patient suddenly developed *tearing chest pain*. There was subcutaneous emphysema in the neck. *Characteristic* crunching sounds were heard. The clicks responsible for the crunch (X) are *unusually musical* and are present in diastole as well as *systole*.

During World War I it was noted (1413) *that in penetrating wounds of the chest in the region of* the heart there might be heard a sound "of the nature of a click which . . . varies from a faint sound heard by careful auscultation to a noise which may be compared *to* that heard in the earpiece of a telephone when the lever is moved up and down. It may be heard sometimes when standing at the foot of the patient's bed. Sometimes the *patient* is conscious of it. . . ." Smith (1413) seems to have *invented* the expression *pericardial knock*.

In 1928 Laster (954) described a 31-year-old *man* who developed *spontaneous pneumothorax*

"As he sat giving the history of his illness, at a distance of some five feet a hollow knocking sound could easily be heard coming from the patient's chest." It was loudest at LLSB in expiration, and *in the upright position, resembled the Korotkoff* sounds in quality, and was mid systolic in timing. Frost and Bing (499) described a case in which the extra sound "most resembled the clicking of the tongue used to urge on a horse." Scadding and Wood (1353), Sharpey Schafer (1380), Edwards and Simpson (414), and others have reported on similar cases. Occurrence of the pneumothorax on the *left* seems to be a constant feature.

Mediastinal emphysema, although occasionally remarked upon by earlier writers including Laennec (see p. 8), was brought to clinicians' attention mainly by Louis Hamman in the late 1930's. The severity of the chest pain, suggesting myocardial infarction, makes the condition of importance to cardiologists. Curious sounds, synchronous with the heart beat and occasionally very loud, are frequent in such cases (Fig. 468). The sound has been compared to the "wadding up of paper" (1581), to "a crunching crackling sound like small chicken bones being crushed" (1209), "the noise that one hears on crumpling a handful of cellophane close to the ear" (952). The sounds are frequently audible at a distance of several feet from the patient. The phonocardiogram (693) in mediastinal emphysema shows a succession of clicks in both systole and diastole (Fig. 468). There is essentially complete lack of pattern to the timing and intensity of the clicks, unlike those of pleuropericardial origin.

On the basis of personal experience in 7 patients and a review of 98 cases of so diagnosed spontaneous mediastinal emphysema reported in the literature, Scott (1370) raises the question of

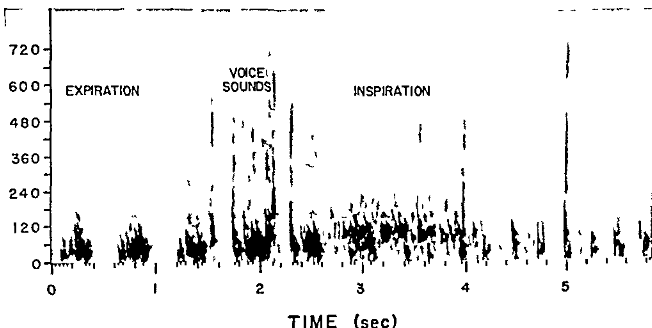


Fig. 469. Systolic murmur caused by pulmonary artery compression.

Here is shown a continuous recording from the pulmonary area of a patient with lymphosarcoma of the mediastinum, on the left in the normal expiratory chest position and on the right in inspiration. A harsh systolic murmur *present in expiration* disappears with *inspiration*. The artifacts in the recording are spoken voice sound and breath sounds. Note the disappearance of the murmur with inspiration. Note further the splitting of the second sound which appears with inspiration. The murmur in this patient is believed to have been the result of pressure of a lymphosarcomatous mass on the pulmonary artery. Inspiration by increasing the *capacity of the chest* removed this compression. The murmur in this case has the typical appearance of an ejection stenosis murmur, which, to be sure, it is. Its peak occurs later in systole than the peak of the ejection steno*sis murmur of aortic origin*, for instance. In some reported cases cardiovascular disease has been simulated by mediastinal tumor. Pulmonary artery compression has been proved to be the cause of the systolic murmur, and the murmur has disappeared after removal of the tumor (499A, 1574A).

whether many of these cases may not in fact be instances of shallow left pneumothorax without mediastinal emphysema. He argues convincingly that the auscultatory phenomena in the two conditions may be identical. Subcutaneous emphysema and x-ray evidence of air in the mediastinum were lacking in many of the cases. On the other hand, in 48 of the 98 reported cases a small left pneumothorax was described. In only four was there a right pneumothorax. Pertinent to this discussion is the mechanism of pneumothorax: whether it arises first as interstitial emphysema, as believed by the Macklin (1014) or through rupture of a subpleural bleb. Chapman (249) has also raised a question of the specificity of Hamman's sign for mediastinal emphysema.

Possibly the circumstances in Hamman's first case (631) are consistent with origin of the adventitious sounds in a shallow left pneumothorax alone rather than in mediastinal emphysema:

> the patient laughingly said he could reproduce the sound. He turned on his left side, shifted about a few moments and suddenly said "There it is." I put my stethoscope over the apex beat of the heart and with each impulse heard the most amazing sound. It is difficult to describe. Crunching is the best adjective I can think of though it is far from apt, especially since crunching has been widely used to describe pleural frictions to which it bears no resemblance. It certainly conveyed the impression of air being churned or squeezed about in the tissues. . . . When the patient turned on his back the sound at once disappeared.

Allen (17) described an unusual case of huge *diaphragmatic hernia* in which the patient, a woman, came for examination complaining of heart trouble . . . because of a disturbing noise synchronous with the heart beat and present intermittently for two years. It was especially bothersome in bed at night and was specifically described by the patient as resembling the splash ing of a water wheel. Greene (591) found that introduction of air into the colon in amounts sufficient to *distend the splenic flexure* would result in a metallic knocking noise, sometimes systolic, sometimes diastolic. Roberts (1283) and Bean (74, 75) have pointed out that splashing or knocking sounds may be audible when the stomach is filled by a proper mixture of gas and fluid and the heart is active. These circumstances may obtain in normal individuals after a large meal, especially if the subject is seated slouched in an upholstered chair, or in a neurotic individual with cardiac hyperactivity and air swallowing it may be the basis of puzzling symptomatology. The *normal individual may be able to abolish the sound by belching*.

Bean (74, 75) describes the observation of one of his associates, Dr. Lewis E. January, who studied a healthy young man with the peculiar ability to make his heart sounds audible to a distance of several inches when he held his mouth open. There was no evidence of heart disease or of abnormality of the lungs, esophagus and upper GI tract. It was suggested that through some anatomical or functional quirk the subject kept the esophagus patulous when the mouth was open, and that the esophagus behaved like a megaphone.

Diaphragmatic flutter (373) is a *condition with* several features suggesting heart disease. Pain in the chest, shoulder and abdomen simulates angina pectoris; it has been called diaphragmatic angina. The patient may complain of palpitation. On auscultation a to-and-fro shuffle like a pericardial *friction* rub or a tick-tock, tapping, swishing or churning sound may be heard. It should be possible to distinguish the sounds from any of cardiovascular origin by the fact that the rhythm is distinct from that of the heart. The adventitious sounds are heard well away from the precordium and over the lower part of the chest. The phenomenon persists in sleep. Diaphragmatic flutter occurs in paroxysms, is often *cryptogenic*, and is rare—much rarer than might be thought from the number of reports in the literature, since one famous patient has been reported at least six times, beginning with William Branch Porter (1220) in 1936 (214, 315, 568, 1543). *Intercostal* flutter is even rarer. Diaphragmatic and intercostal flutter are varieties of respiratory tic.

PREGNANCY

The murmurs of pre-existing heart disease, congenital or rheumatic, for example, tend to be altered so markedly during pregnancy that evaluation of their functional significance may be difficult and risky. On the other hand, pregnancy affords an excellent opportunity for the detection of heart disease (2024, 3). Pregnancy is the only

occasion for many young women to have a physical examination. 2) By accentuating diastolic and systolic murmurs pregnancy will facilitate the diagnosis of heart disease, although precise evaluation of its significance may have to await delivery.

A systolic murmur in the pulmonary area occurs commonly in pregnant women probably on the basis of increased stroke volume and possibly displacement of the heart. Systolic clicks may develop on the latter basis just as they may be heard in massive ascites—displacement of the heart may cause it to press on the anterior rib cage with movement of costochondral and/or chondrosternal joints.

Burwell and Metcalfe (202A) have, contrary to the stated experience of others (191A), never heard a basilar diastolic murmur in pregnancy in a patient who could be considered free of heart disease. A murmur of the Carey Coombs type at the apex probably does not occur in physiologic pregnancy. For it to occur unusual anemia, rheumatic carditis, or a congenital malformation must be present.

In late pregnancy and early after parturition a mammary souffle (see p. 233) may be heard over both breasts, usually at the upper border. The murmur may be only systolic, specifically late systolic, or may be continuous. The facts that it is obliterated by pressure on the stethoscope and that it is confined to late systole, when combined with an acquaintance with the phenomenon, will usually suffice for identifying the murmur.

Venous hums occur commonly. Pistol shots over peripheral arteries are not uncommon.

The uterine souffle, fetal heart sound and other sonic phenomena related to the uterus and its contents have been described on pp. 233 and 255.

SECTION V

Physiologic, Pharmacologic, Physical and Other Procedures Employed in the Investigation of Cardiovascular Sound in Man

CHAPTER 22

Procedures for Study of Cardiovascular Sound in Man

With a Section on Quantification

As was noted on p. 101, phonocardiography and the study of cardiovascular sound in general acquire most significance when quantitation is possible and when they are elevated from the realm of pure observation to that of an experimental science by the designed use of various test procedures. It is the purpose of this section to review and analyze what has been done in this area.

RESPIRATION (173, 823). The normal inspiratory splitting of S_2 (p. 159), the paradoxical splitting (i.e., splitting in expiration) which occurs under certain circumstances, the exaggeration of the murmurs of tricuspid stenosis and regurgitation during inspiration (p. 324) are useful in the study of patients.

Maronde *et al.* (1043) have pointed out the existence of a paradoxical paradoxical pulse in left ventricular failure. The pulse increases in inspiration rather than decreasing as is usually the case. The phenomenon disappeared with compensation. Parallel changes in the intensity of murmurs of aortic and mitral valve disease might be expected although the magnitude of the change is probably slight.

THE VALSALVA MANEUVER. During straining the increase in intrathoracic pressure causes an immediate reduction in venous return to the heart. The output of the right heart decreases, the pulmonary reservoir is depleted, and the output of the left heart falls. With relaxation the venous blood which was dammed back peripherally rushes into the heart. The pulsations in the pulmonary artery, previously reduced in amplitude, become vigorous at once according to information supplied by electrokymography (772). The output of the left heart and aortic pulsation become maximal only after refilling of the pulmonary reservoir.

Zinsser and Kay (1606) found that the behavior of murmurs after the release of Valsalva type straining is useful in distinguishing right sided and left sided lesions. Most murmurs were diminished sharply by straining. In the case of right sided lesions the murmur returned promptly with cessation of straining whereas a delay was observed with left sided lesions. The murmur of pulmonary AV fistula returned early. Functional pulmonary systolic murmurs behaved like right sided lesions.

Mitral stenosis and patent ductus arteriosus behaved in a modified but characteristic manner. The mitral opening snap often disappeared with straining and returned a few cycles after release of straining. The early passive portion of the diastolic murmur behaved in a manner typical of left sided lesions. The presystolic murmur varied more as a function of heart rate than of the changes in volume of flow which were presumed to accompany straining. Because of the tachycardia during straining the presystolic murmur might be increased paradoxically in that period and be decreased with the bradycardia of the post strain period.

The murmur of patent ductus arteriosus was moderately reduced by straining. Interestingly in the early post strain period further reduction in the murmur usually occurred. The increased pulmonary flow at that time apparently reduced flow in the shunt.

As one might expect, P_2 became split in the

early post strain period Zinsser and Kay (1606) thought the first element of the split was pulmonary closure sound—a view difficult to reconcile with other information—mainly because this element seemed to be accentuated early and the second element accentuated late in the post strain period

In experimental murmur producing lesions in dogs, Bertrand and collaborators (96) confirmed the clinical observations However, observations on the Valsalva maneuver in heart failure (577, 807, 1379) leave some doubt as to the validity of the test when central blood volume is increased Specifically, in heart failure no change in pulse pressure and no post straining "overshoot" was observed No change in murmurs can therefore, be anticipated Practically speaking, patients in heart failure are often too dyspneic or too sick and weak (1605) to perform the test adequately Goldberg Elisberg and Katz (563) found that in mitral stenosis of only acoustic significance the response to the Valsalva maneuver was identical to that in normal an 'overshoot' of blood pressure and bradycardia in the post strain period In severe mitral stenosis these normal phenomena were abolished (590) With straining there is a damming up of blood in the systemic venous system With release of straining venous return to the heart increases markedly through release of this dam assisted by the hyperpnea of the post strain period The increased cardiac output accounts for the "overshoot" in blood pressure which in turn produces the bradycardia by reflex mechanisms In severe mitral stenosis mitral flow cannot increase sufficiently to produce these changes In those few healthy individuals who for some reason can maintain blood pressure during the Valsalva autonomic block with tetraethyl ammonium chloride (590) will return the pattern to "normal" whereas the patients with significant mitral stenosis maintain brachial pressure during the Valsalva despite TEAC

With the Valsalva maneuver Lee and Gimlette (867) produced partial reversion of the shunt in ASD The effect on splitting of the second sound should be investigated Reversal of the left to right shunt occurs during the post straining period During straining the left to right shunt may be increased

TOURNIQUETS Leonard and colleagues (876) have demonstrated the usefulness of the application of tourniquets in the study of gallops The effect of impounding of blood in the extremities (819) may have usefulness in connection with other sonic phenomena

EPINEPHRINE, NOREPINEPHRINE, AND RELATED SUBSTANCES Epinephrine has been shown to abbreviate the isometric contraction period of the ventricle (801, 802) The pre isometric period is probably shortened also The result is that S_1 is accentuated by the more rapid closure of the AV valves and becomes snappier through the addition of components of higher frequency The QS_1 interval is reduced These phenomena may occur with epinephrine in dosages insufficient to affect blood pressure Stimulation of the sympathetic nerves to the heart (or section of the vagi) has a similar effect (1551)

With larger doses of epinephrine the second sound likewise becomes accentuated The effects produced and the changes observed in the heart sounds are related to both the greater and the lesser circulations

With epinephrine the Korotkoff sounds are intensified (275) Lewis and Hewlett (899) concluded by comparing the effects of nitroglycerine that the effect was the result mainly of increase in the stroke output

Because of increased velocity of blood flow, any systolic murmur—including the functional pulmonary systolic murmur of young subjects—is likely to be exaggerated by adrenaline Holldack and Mielke (705) heard and recorded a diastolic murmur in the third and fourth left interspace after adrenaline It had the timing of a mitral diastolic but the quality of an aortic They thought it was produced by the pulmonic valve

Posterior pituitary extract was shown by Wiggers (1550 1552) to increase systemic arterial pressure at the same time producing a fall in pulmonary arterial pressure He further observed in dog (1548) the expected changes in A and P Posterior pituitary extract has little or no effect on blood pressure in man (588A) however

Amyl nitrite has been suggested for eliciting the murmur of mitral stenosis (888) for identifying functional aortic insufficiency (526), and for differentiating between aortic stenosis and mitral

regurgitation (58). Carvin (326) claimed that 14 of 200 autopsied cases of hypertension showed minimal aortic regurgitation of "relative" type. Amyl nitrite abolished the murmur by *reducing diastolic pressure*. Barlow and Shillingford (58) suggested amyl nitrite for differentiating regurgitant from ejection stenotic murmurs. Amyl nitrite reduces diastolic pressure and effects a decrease in a *mitral regurgitant* murmur. It increases cardiac output and increases an ejection stenosis murmur (e.g., that of aortic stenosis).

The breathing of mixtures containing *reduced amounts of oxygen* is accompanied by increases in pulmonary artery pressure (1121). The effect on murmurs such as that of patent ductus arteriosus or pulmonary regurgitation may be diagnostically helpful. Mannheimer (1032) administered 9 per cent oxygen mixture to four patients with patent ducts, but it is not clear from his report what happened to the murmur.

POSITION. The head-down position can be used to accentuate atrial heart sounds (433). The recumbent posture is the best for detecting the physiologic third heart sound, especially *primo decubitus*, the first assumption of the recumbent posture. The left lateral decubitus and sitting leaned forward postures are usually *optimum for* audibility of the murmurs of mitral stenosis and aortic regurgitation, respectively. The *knee-chest* position is sometimes useful in hearing the murmur of aortic regurgitation. To detect a pericardial friction rub it may be necessary to place the patient in many different positions.

In 1902 Gordon (573) found that the venous hum disappeared in the recumbent position but hemic murmur *and the* murmurs of *tricuspid regurgitation*, mitral regurgitation and aortic stenosis increased. The murmur of aortic regurgitation showed little change.

CARDIAC CATHETERIZATION (1377). Aside from important contribution *toward* establishing the diagnosis in many instances, cardiac catheterization provides or may eventually provide four main types of information *useful to the study of* cardiovascular sound: (1) volume of blood flow including differences in *stroke volume of the two* ventricles in the case of shunts; (2) intravascular pressures (gradients across *heart* valves and septal defects; the time course of phasic pressure changes *for correlation with the shape of murmurs, timing of mechanical events for identification of nature of transient, e.g.*, an opening sound of the arterial valve); (3) the velocity of blood flow (600, 601, 1132); (4) in combination with appropriate injection of a dye or other indicator and sampling from other sites, cardiac catheterization may eventually permit quantitation of valvular regurgitation. In addition, of course, intracardiac phonocardiography with a catheter microphone can be combined with conventional catheterization method.

The pulmonary capillary ("wedge") *pressure* is useful in differentiating functional *from organic* mitral stenosis. In a *patient* with aortic regurgitation due to rheumatism, Wade, *Ross*, *ch* and Werko (1498) were *able to identify the* Austin Flint apical diastolic murmur as an Austin Flint by the fact that *pulmonary capillary* pressure was *normal* both at rest and with exercise. By the finding of an elevated PC pressure in a patient with *sickle cell anemia* I (1070) was able to establish that the apical diastolic murmur resulted from organic mitral stenosis, a fact confirmed by autopsy.

Ever since the method was first introduced a *century* ago by Chauveau in collaboration with Marey and others (e.g., p. 12), the timing of events in both sides of the heart by simultaneous recordings of intracardiovascular pressure pulses and of the heart sound has been important in elucidating the origin of the sound. Right heart *catheterization* in man—and more recently left heart catheterization—have contributed precise information *on* the time sequence (166, 170, 276, 370, 373, 372).

The recording of heart sound during cardiac catheterization ensures that the correlation of cardiovascular sound with hemodynamic are reliable. Changing hemodynamics by measures such as exercise, breathing of reduced oxygen mixtures, *pharmacologic agents* is now customarily *practiced in* combination with cardiac catheterization. Correlation with the changes in heart sound and murmurs should be investigated *more extensively*.

Pressure recording at cardiac catheterization *of both sides of the* heart or at operation by puncture of the *proper vessel* or chambers can be useful in describing the pressure gradient across

stenotic orifices, regurgitant orifices, septal defects, etc Gordon and his co workers (570, 571) have demonstrated the usefulness of simultaneously recorded pressures using equisensitive transducers and the same base line in the recordings It is entirely feasible to have a continuous recording of pressure differential, that is, the difference in pressure sensed by the two transducers, or, of course, differential pressure can be calculated Finally, when sound recordings are made simultaneously, the pressure recordings permit identification of various elements of the heart sounds and investigation of the relation between pressure gradient and murmur

PHYSICAL EXERTION Exercise is used more in connection with eliciting a mitral diastolic murmur of mitral stenosis than in any other single connection Frequently mitral stenosis of minor grade is missed when a patient is examined while lying quietly in hospital Even "sit ups" may be inadequate for demonstrating it In the same patient, ambulatory under the conditions of an outpatient clinic, the murmur of mitral stenosis may be very evident A physiologic third heart sound is likely to be brought out by exercise of the legs in the recumbent posture Lian and Hubert (927) found that exercise would bring out a presystolic gallop in persons such as hypertensives who did not previously demonstrate it

Borter and Muller (138) found that with exertion the QS interval was shortened more than the QT interval of the electrocardiogram The QT interval was abbreviated and S_1 was increased in intensity

RADIOLOGIC METHODS FOR STUDYING TURBULENCE AND (INDIRECTLY) MURMURS This is an approach in which Dotter (365) is pioneering Occasionally—in the early phases of filling—turbulence may be indicated in angiocardiograms in patients with certain lesions Usually, however there is a blurring of detail comparable in the vivid analogy drawn by Dotter (365) to the loss of the detail of movement of individual cars down a city street in a night photograph made with a long exposure

Necessary for the radiologic demonstration and analysis of turbulence are (1) very brief x ray exposures, and (2) a flocculent radio opaque medium Both requirements present practical difficulties which have not yet been overcome Dotter (364) states "When it becomes roentgenologically possible to visualize turbulence, the stethoscope may indeed be administered a 'mortal blow'" Although it is difficult to imagine any diagnostic method involving an injection which would combine the informative and safe feature of the stethoscope, this excessively enthusiastic statement at least indicates a technique which can contribute to our understanding of the generation of sound in the cardiovascular system

THE QUANTIFICATION OF REGURGITATION Rapid advances are being made in the development of methods for quantitative estimation of the volume of aortic and mitral regurgitation (167) In the future correlations of the volume of regurgitation (along with other parameters such as pressure gradient and geometry of the valve lesion) with the characteristics of the murmur should provide information of theoretical interest and practical value

QUANTIFICATION IN CARDIOVASCULAR SOUND

The three parameters of cardiovascular sound have been described in considerable detail but mainly in qualitative terms The temporal dimension is described with reference to the phase of the cardiac cycle with regard to the approximate relationship of one element to another, and with respect to duration of each element Frequency is indicated by rough adjectives qualifying pitch Intensity is graded by approximate systems adequate for most clinical purposes such as the system of Freeman and Levine (481) The largest portion of this monograph has been occupied with a review of the qualitative description of cardiovascular sound in health and disease Phonocardiography attains its highest precision and greatest physiologic usefulness when quantitation is applied in each of the three parameters of heart sounds It is the purpose of this section to review what has been accomplished in this domain

The dimension of frequency can be dismissed briefly since the spectral phonocardiogram accomplishes quantification In fact such a wealth of information is provided by the spectral phonocardiogram that a problem is created in the handling of the mass of data in such a way that one

record can be compared with another How for example is the average frequency content of the first heart sound in a series of normal subject to be determined from the spectral phonocardiogram? Furthermore relating frequency to anatomy and hemodynamics encounters complexities which are not easily unraveled

Measurements of intervals between two sonic elements or between a sonic element and another cardiovascular event and the relating of these elements to independently measured cardiovascular parameters have represented a most admirable demonstration of the use of phonocardiography as a physiologic tool The following is a list of certain of the quantitative studies of the temporal dimension of cardiovascular sound

1 The relation of the Q 1 (onset of Q wave of EKG to onset of rapid vibrations of S_1) interval to left atrial pressure use of the Q 1 interval as an index of the grade of mitral stenosis (778)

2 The relation of the interval between S_2 and the opening snap to the left atrial pressure use of the S–OS interval as an index of the grade of mitral stenosis (64 66)

3 The relation of the interval between the aortic and pulmonic components of S_2 (AP interval) to the level of right ventricular pressure and therefore the severity of pulmonary stenosis (874)

4 Relation of the QK interval (between the Q wave of the EKG and the onset of the Korotkoff sound) to the preceding RR interval use of this relationship as evidence that stroke output influences pulse wave velocity (1296)

The intensity parameter of cardiovascular sound is possibly the most difficult to quantify The inaccessibility of the generator and the variability and complexity of the surrounding transmitting tissues are part of the problem Since cardiovascular sounds of equal net intensity may vary widely in frequency composition measurements which do not take this fact into account are of somewhat limited usefulness

THE MEASUREMENT OF INTENSITY IN CARDIOVASCULAR SOUND The problem and the several approaches which have been employed are discussed elsewhere One method used in the measurement of noises of other types is that described by Churcher and King (264 265) The noise to be quantitated is compared with a reference tone at 1000 c p s The intensity of the reference tone is changed until the two match The procedure can be done by putting the test noise in one ear and the reference tone in the other or by putting both in both earphones but playing them alternately The method is comparable to the use of a comparator in colorimetric chemical determination Contrary to what one might think *it is* possible to match wide band noises to pure tone with satisfactory reproducibility The transients such as the first and second heart sound are probably measured only with difficulty and wide approximation by this method but murmurs can be quantitated

Another and more satisfactory way to measure intensity is to listen with an electronic stethoscope and adjust a calibrated attenuator until the intensity of the sound to be measured disappears (879 1108 1319 1352)

SECTION VI

Cardiovascular Sound in Animals

CHAPTER 23

Cardiovascular Sound in Animals[1]

TECHNIQUES

To obtain proper electrocardiograms for correlative purposes plate electrodes of the conventional type (with electrode paste) must sometimes be replaced by venopuncture needles passed through the skin and connected to the appropriate lead wires of the EKG machine. Application of the microphone to the chest may be better accomplished by wetting down the hair or even by shaving it. A larger chest bell than that ordinarily employed may be desirable.

DISCUSSION

HORSE. The important role of the horse in the elucidation of cardiovascular sound has been commented on (p. 42) in the discussion of the work of Chauveau. Electrocardiographic studies were performed by Einthoven (417), Kahn (763), Norr (1146), Neumann-Kleinpaul and Steffan (1141) and others. The heart rate of the horse is usually between 32 and 50. The P wave of the EKG is interestingly complex; it is usually double (994) and may be even more complex with an additional initiating wave attributed by Norr (1146) to the sino-atrial node and two late waves which may represent T waves of the two components of the P wave. The PR interval is usually of the order of 0.30 sec.

Luisada and colleagues (994) found *no atrial sound* except in old or sick horses. When present it began 0.20–0.30 second after the beginning of P. The interval between the heart sounds was usually 0.52 to 0.62 sec. In some records of large horses vibrations of low frequency were seen beginning 0.10 sec after S occurring therefore during and possibly related to rapid ventricular filling. These vibrations lasted 0.15–0.25 sec. Rarely was a third heart sound recorded.

Mitral systolic murmurs seem to occur rather frequently in the horse (319, 975). Mitral regurgitation was apparently the cause in Detweiler's (319) 11-year-old gelding which also had atrial fibrillation. Clinical signs of tricuspid regurgitation and clinical and autopsy manifestations of congestive heart failure were described. A prolonged third heart sound was present.

In two donkeys Luisada, Weisz and Hantman (994) found a third heart sound.

A typical continuous murmur of physiologic patency of the ductus arteriosus has been described in the newborn foal (184).

CATTLE. The heart rate varies from 30 to 60 for the bull, 60 to 80 for the cow and 110 to 134 for the calf (380, 1146).

In the record of one bull the second sound was periodically split apparently following the respiratory phases (994).

As in the foal the continuous murmur of PDA has been recorded in the newborn calf (184).

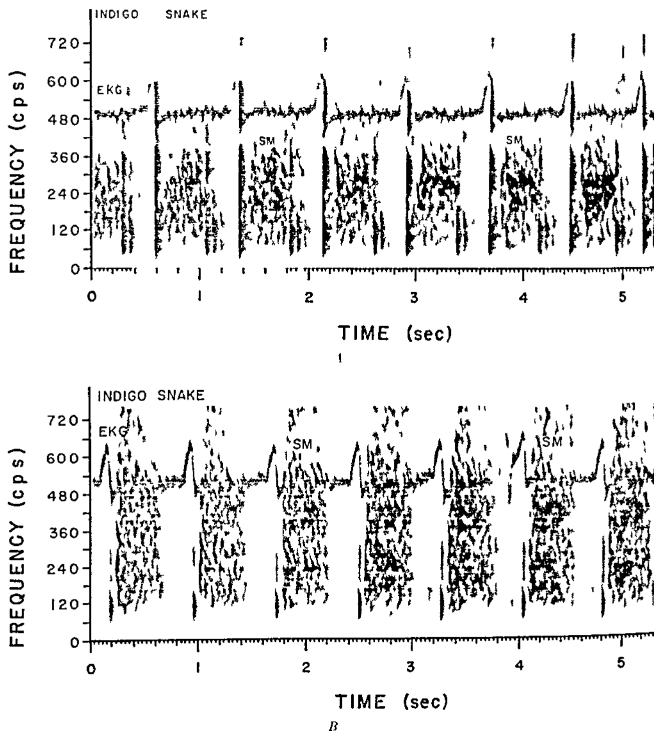
HOG. The heart rate is highly variable apparently depending on age and size. Vibrations due to atrial systole have been identified (994).

SHEEP. Atrial sounds were recorded by Luisada, Weisz and Hantman (994).

DOG. The dog has, of course, been studied extensively in connection with cardiovascular investigations of many types (1399). See Figures 129 and 131 for examples.

RABBIT AND GUINEA PIG. The heart rate is of the order of 190 for the rabbit and [illegible]00 for the guinea pig. Respiratory rate in the rabbit is often about half the heart rate so that inspiratory and expiratory sounds are easily confused with the heart sounds or with murmurs. In the rabbit as

[1] The assistance of Mr. Arthur P. Watson, director of the Baltimore Zoo, is gratefully acknowledged.



B

Fig 470 Indigo snake

Recorded from 35th ventral segment in area of apex beat and maximum QRS of the electrocardiogram in 7 foot snake

in the rat and man the second sound occurs at the end of the T wave (585)

RAT AND MOUSE The heart rate in the rat is 400 to 500 and in the mouse 500 to 700 per minute

OTHER ANIMALS In the non hibernating hedgehog Johansson (748) and in the kangaroo Sporri (1426) found a situation opposite to the Hegglin syndrome (p 119) In these animals S occurs appreciably later than the T wave indicating that mechanical systole is longer than electrical systole The elephant's physiology has been studied from several points of view (84) including a description

of the low heart rate of about 35 beats per minute Apparently phonocardiograms have not been made

We have recorded the heart sounds in frogs, turtles, alligators and snakes. Figure 470 illustrates the findings in 6 to 7 foot snakes (indigo snake *Drymarchon corais couperi* and rock python *Python sebae*) and in 9 foot boa constrictors (*Constrictor constrictor imperator*). The QRS of the electrocardiogram, electrical systole and mechanical systole as indicated by the interval between the first and second sounds are prolonged consistent with the expected findings in a cold blooded animal. The snake has an interventricular septal defect and has three arterial trunks arising separately from the incompletely divided ventricle pulmonary artery and two aortae (81A) (476A) (476B). Each arterial trunk has a bicuspid valve at its mouth (1248A).

The three elements in the part of the cardiac cycle expected for S (Fig. 470A) may be produced by closure of the three arterial valves. The systolic murmur is not as we first thought generated at the ventricular septal defect or elsewhere in the heart and great vessels. It is generated artifactually at the skin surface by rubbing of scales between the apex beat and the microphone.

GENERAL COMMENT

The duration of the heart sounds diminishes and the frequency span of the heart sounds increases with diminishing size of the animal and increasing heart rate.

SECTION VII

Respiratory Sound (Lung Sounds)

CHAPTER 21

Lung Sounds

PERCUSSION NOTES

The characteristics of tympanitic, resonant, and dull percussion notes are compared in Figure 471. The three have in common a central core which is the sound of impact of the percussing finger on the pleximeter finger. This impact sound constitutes a considerable part of what one hears in the percussion note. In the case of the completely flat note there is little else because the blow is not able to set the structures under the percussing finger into vibration to any significant extent. In the case of the resonant note vibrations maximal in the range of the natural frequency of the thorax are superimposed on and follow the impact sound. The natural frequency of the subject's thorax (a normal young male adult) is seen to be approximately 140 cycles per second (c.p.s.).

Comparing the resonant and the dull notes, it appears that the former is louder and longer than the dull note as a result of the additional forced vibrations of the normal air filled thorax. The dull note is on the average (and in the way it impresses the ear) of a higher pitch. This is not principally because *vibrations* of higher frequency have been excited, but because the average pitch of the dull note is not weighted by *the low* frequency vibrations of the thorax. It is true that in the relatively dull note of pulmonary consolidation *vibrations* of higher frequency and lesser intensity are incited in the pathologic tissue which has a higher natural frequency.

Comparison of the tympanitic note (e.g., over the stomach bubble) with the other notes *reveals* that the main difference is a more musical quality which is reflected in these displays by the presence of more clearly defined harmonics. There is a distinct fundamental at 180 c.p.s. and the second harmonic at 360 c.p.s. is equally well defined.

EARLY STUDIES OF PERCUSSION NOTES. The lesser intensity of the dull percussion note as compared with the resonant was demonstrated by Selling (1873) in Friedrich Muller's laboratory by a simple but ingenious method. He compared the maximum distance at which each type of note could be heard when produced by a physician percussing over the lung and over the thigh with equal force. He found that the resonant note is audible approximately five times as far as the dull note. Since intensity is inversely proportional to the square of the distance, it can be concluded that the healthy lung encased in the thoracic cage is approximately twenty five times more resonant than the muscle mass of the thigh. Selling and Scripture also recorded percussion notes on phonograph cylinders of the Edison type and demonstrated by microscopy that the cut produced by the resonant note was considerably deeper than that produced by the dull note. In an even more definitive experiment Selling and Edelmann measured the intensity of these notes by means of the Einthoven string galvanometer and showed that the dull percussion note was of considerably less intensity. The greater duration of the resonant note was likewise *demonstrated by* Selling's studies by both the phonographic and the Einthoven galvanometric method.

The timbre of the resonant or vesicular percussion note has been compared with the timbre of the note produced by striking a loaf of bread covered by its crust—the air cells of the bread are analogous to the alveoli. Pratt (1275) observed that when milk is stirred as it is brought to a boil the stirring produces a resonant note when the boiling point is reached. Again the boiling milk contains *innumerable* tiny bubbles filled with vapor under pressure and separated by

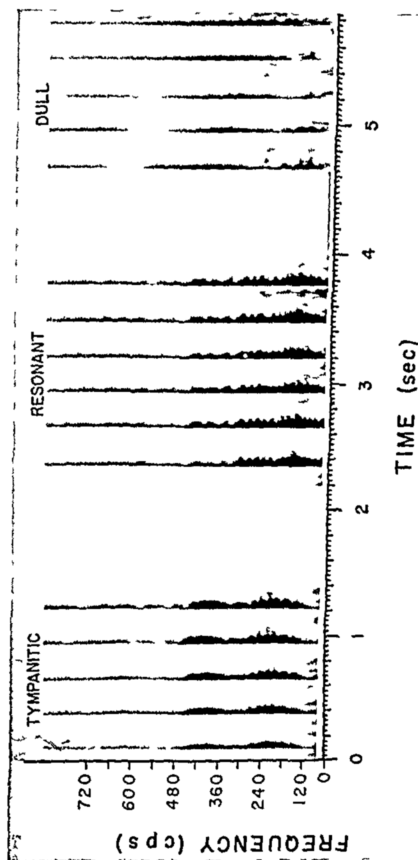


Fig 471 Percussion notes

liquid partitions endowed with a certain amount of elasticity Geigel (533) compared the tympanitic percussion note with that produced by percussing a glass cylinder filled with very frothy beer

Placing the pitch of percussion notes on the musical scale is difficult because they are more nearly noises than musical notes Nevertheless an average pitch can be determined particularly by comparison with the pitch of other notes just as each of the kettledrums of an orchestra emits a noise of distinct average pitch and a melody can be played on a series of glass tumblers Auenbrugger and Skoda noted the variation in pitch of different resonant percussion notes and Austin Flint (466) emphasized this fundamental acoustic characteristic Gerhardt in 1876 (541) and Selling in 1907 (1375) used Helmholtz resonators for studying percussion sound Selling found it necessary to use cylindro-conical resonators as much as 2 0 meters in length to study the lowest pitched of the percussion notes These studies and those of Martini (1044) and of Rist (1275) demonstrated a series of tones ranging from a pitch corresponding to F below low C (approximately 80 c p s) in the musical scale up to C above high C (approximately 800 c p s) and depending on the volume of the thorax (see below) Martini (1044) placed the fundamental of the natural frequency of the child s lung at 170 c p s of consolidated lung at 140 to 190 c p s , of normal adult lung at 100 to 120 c p s and of emphysematous lung at 70 to 90 c p s

The character of the plessimeter impact sound is determined by the nature of the plessimeter The ivory and wooden plessimeters of an earlier period were shrill and noisy because they were stiff In digito-digital percussion now used almost exclusively the impact note is less striking yet clearly discernible It is this plessimeter impact note which is heard predominantly in the dull percussion note and exclusively in the absolutely flat percussion note According to Selling a plessimeter of soft rubber is almost silent

Another older method by which the frequency composition of percussion notes was studied many years ago by Cartex (241) is that which involved photographing manometer flames In this method devised by Koenig (see Miller (1103)) in 1862 the gas pressure to the flame is modulated by a delicate membrane which vibrates with the sound be studied Variations in the height of the flame are what are photographed on moving film to produce a record of the sound vibrations Selling applied Scripture s method to the problem (see Miller (1103)) Oscillographic like tracings were made by a machine which took the vibrations of the gramophone disk and after passage through a system of delicate compound levers marked them on the smoked paper of a kymograph

Montgomery (1111) states that the fundamental if such it can be called of vesicular (resonant) percussion and of normal vesicular breathing is approximately 108 c p s a close match with 128 c p s the fundamental of man s voice The fundamental of a woman s voice is closer to 256 c p s

It is a general experience that tympanitic percussion notes are more musical than the resonant ones their pitch is more easily placed in a musical scale Selling s string galvanometer recordings of tympanitic notes (1375) displayed more regular vibrations than were found in resonant notes which produced complicated oscillograms The same finding was made by May and Lindemann (reported by Fahr (449)) who used an arduous but ingenious method They obtained an artificial membrane by plunging a metal ring into a soapy solution This delicate membrane vibrated in a highly sensitive manner with sound vibrations and when so placed that it reflected an image on moving photographic film it inscribed an oscillogram of the sound

THE NATURAL FREQUENCY OF THE THORAX It is clear from the previous exposition that the thorax has a natural frequency[1] which is demonstrated by an analysis of percussion notes The natural frequency in general varies inversely with the volume of the thorax When a subject sings a low note the hand placed on the chest will feel the vocal vibration the thorax behaves like a Helmholtz resonator If the subject sings

[1] Natural specific or intrinsic vibrational frequency depends on the tendency of an elastic structure when deformed from its position of rest or equilibrium to return to that position The character of the structure s vibration during the period it is resuming its position of rest is determined by its modulus of elasticity Frequency is directly proportional to density and inversely proportional to volume

an ascending scale, the vocal thrill, palpated on the surface of the chest, will become weaker and disappear. The threshold is higher when the singer is in expiration. The matching of the pitch of the voice to the volume of the thorax is a factor determining whether vocal resonance is present or not. Vocal resonance is the rule in men because of good match. In women vocal resonance is rare because, although the natural frequency of the thorax is higher, the voice is even more shrill than the thorax can match. On the other hand, infants and children are likely to show conspicuous vocal resonance because of having close to ideal match of their more shrill voices to the higher natural frequency of the thorax.

In the strict sense, the thorax is a double coupled system comprising, on one hand, the thoracic wall—particularly the rib cage—and, on the other, the lungs. The natural frequency of the stiffer chest wall is somewhat higher than that of the enclosed structures.

The natural frequency of the thorax is an important factor in the manner in which murmurs are altered in quality in transmission to various parts of the chest. It has been noted that those frequency components of a heart murmur which are in the same general range as the natural frequency of the thorax tend to be transmitted best to various areas of the chest (see p. 150). An experiment comparable with this one of nature is *to place a series of tuning forks one after another* on the anterior portion of the chest and by palpation gauge the extent to which these vibrations of pure pitch are transmitted to the back. (In actuality, satisfactory results from this experiment (1099) are vitiated by circumferential conduction of the vibrations in the rib cage.) Disease processes in the lung and pleura may have the result that the thorax, or a part thereof, resonates differently from normal. The lower pitched components are diminished in intensity and one says that the percussion note is higher pitched. This conclusion is due in part to the fact that the more shrill plessimeter impact note is left to impress the auditory perception to a greater extent. To some extent, also, the higher pitched note is the result of a higher natural frequency of resonation of the diseased thorax. Alterations in this frequency of resonation are important factors in the genesis of "E to A change," change in breath sounds, and other phenomena discussed below.

BREATH SOUNDS

The character of the breath sound, as detected by stethoscope or microphone at the surface of the thorax, is determined to a considerable extent by the resonating and sound-conducting properties of the lung tissue between the larger airways and the point of detection. The average pitch of normal vesicular breath sounds varies in the same manner as the percussion notes, being progressively lower in infants, adult females, adult males, and patients with emphysema.

Diagnostic changes in breath sounds involve principally the expiratory phase. The stethoscopist should pay particular heed to the character of this phase.

Normal vesicular breath sounds are demonstrated in Figure 472. Expiration is relatively noiseless. Electrical interference at 60 c.p.s. is present.

The inspiratory "murmur" or normal vesicular breath sound has its origin at the alveoli (189) and is the result of turbulence created when air currents spread out into the myriads of air sacs. It is well heard at the surface of the chest because it is generated at close hand. Movement of air in the larger airways is not necessary for its production. One variety of cardiopulmonary murmur *is of fundamentally the same origin and has the* same character as the vesicular sound (see p. 225). Other evidence for the vesicular origin of the vesicular inspiration sound (note the appropriateness of the terminology) is provided by the auscultation of eventerated lungs (189) and of hernia of the lung (1363). The expiratory sound, on the other hand, has its origin at bifurcations in the larger airways by a mechanism comparable to the labial pipe of the church organ (1044). Because of the porous sound-absorbing intervening lung tissue it is not surprising that the expiratory sound normally should be poorly heard at the surface of the chest.

In the breath sounds recorded from the immediate vicinity of the trachea (Fig. 473) the properties of the thorax are not the major controlling factors. The pitch of the sounds which are actually noise in inspiration and expiration is

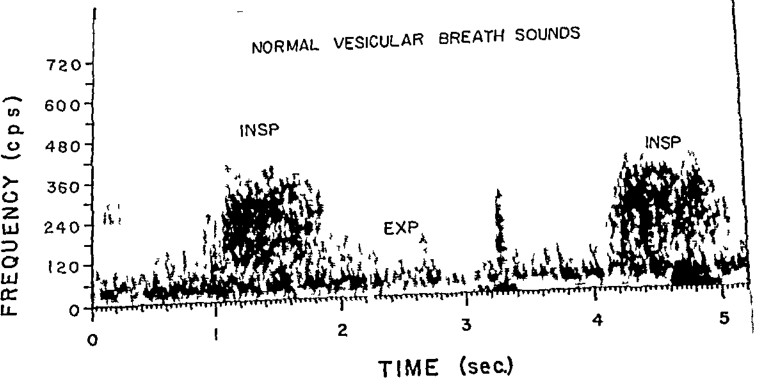


Fig. 4. Normal vesicular breath sounds.

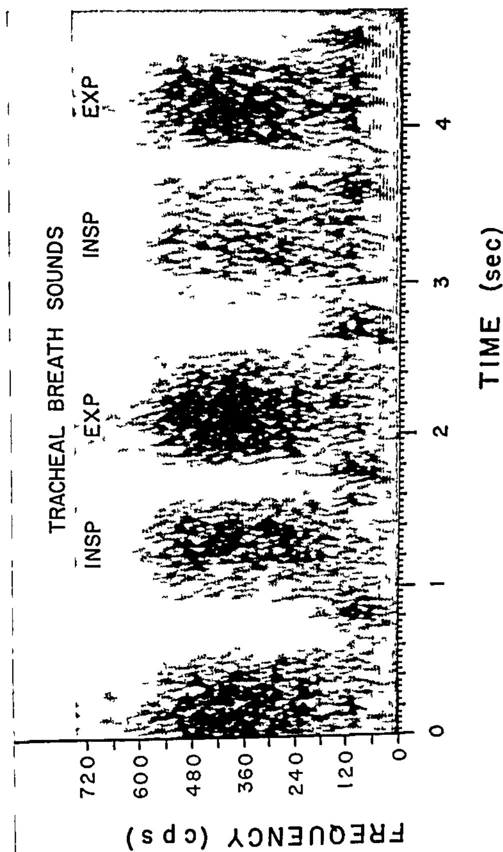


Fig 473 Tracheal breath sounds

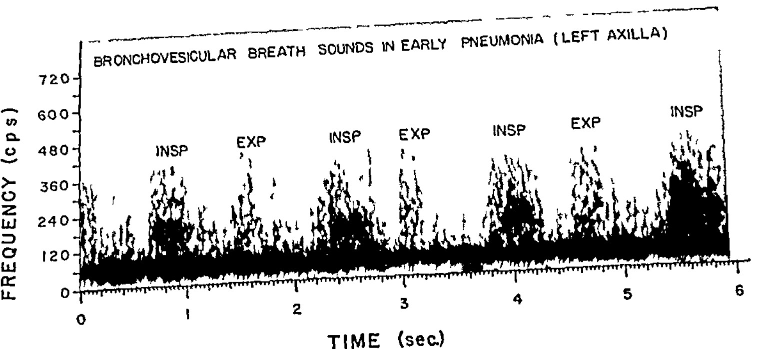


Fig. 471 Bronchovesicular breath sounds in early pneumococcal pneumonia

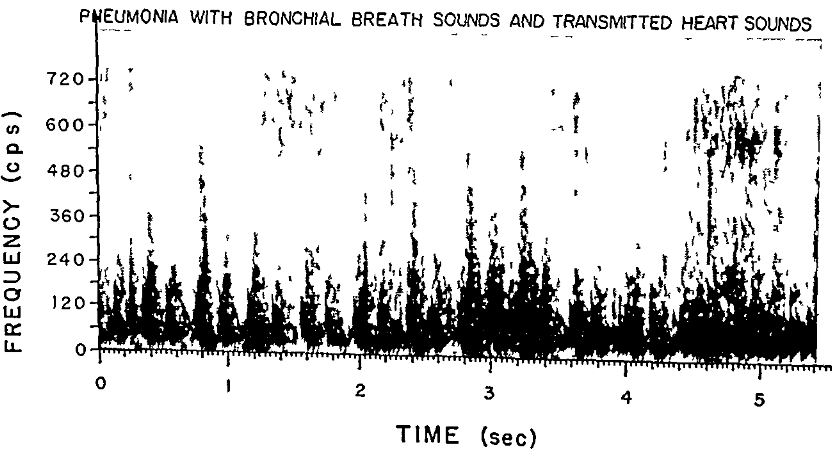


Fig. 475 Bronchial breath sounds (at frequency of 300 c p s up) and transmitted heart sounds in case of pneumonia of the left lower lobe

roughly the same but the expiratory sound is appreciably louder. The writer has observed *variation in the frequency level of the "center"* of the tracheal breath sound. These variations appear to depend upon the dimensions of the trachea, *both length and caliber.* As might be *expected the average pitch of the tracheal sound is lower in persons* who appear to have larger tracheas and vice versa.

When pneumonia produces changes in the sound-conducting properties of lung tissue, expiration which is ordinarily relatively noiseless becomes audible and characteristic bronchovesicular respiration develops (Figure 474). Again electrical interference at 60 c.p.s. is present as well as *some at 120 and 180 c.p.s.*) It may be noted that with partial consolidation it is still only the lower pitched portion of the tracheobronchial sound which are heard at the surface of the thorax. At this stage also *the vesicular inspiratory* sound is likely to be diminished, which is presumably the case in Figure 475, since many *of the alveoli are not functioning because of the exudate.*

Further consolidation of pulmonary tissue such as occurs in *later* stages of pneumococcal pneumonia results in the audibility at the surface of the chest of the vibrations produced in the tracheobronchial tree. In experiments which are now classical, Martini *and* Mueller (101a) de*termined* the *natural vibrational* characteristics of the tracheobronchial tree at its several levels (see graphic representation in reference 1a). These studies indicate that sound produced by *the* mechanism of the labial pipe at the levels of the tracheobronchial tree where the diameter of the tubes is 3 to 10 mm in diameter, i.e., any point below the bifurcation of the trachea, will have a frequency in excess of 1,000 cycles per second. Consolidated lung tissue because of its better conducting properties permit the detection of these vibrations at the surface of the chest. The result is so-called bronchial or tubular breath sound. These are demonstrated in Figure 475. In the example demonstrated an additional feature *of* interest is the presence of heart sounds confined largely to the frequency range below 240 c.p.s. Because the pneumonia in this case was in the left lower lobe the heart sounds were transmitted

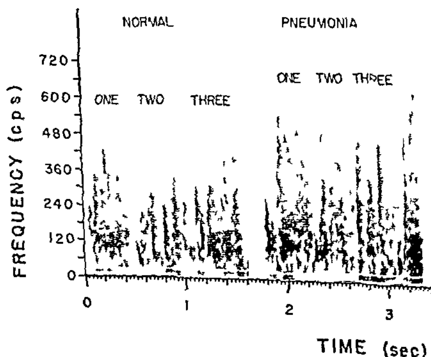


FIG. 476. Augmented whispered sounds in early pneumonia. The change parallels the development of bronchovesicular breath sound. The accentuation *involves both intensity and frequency span.*

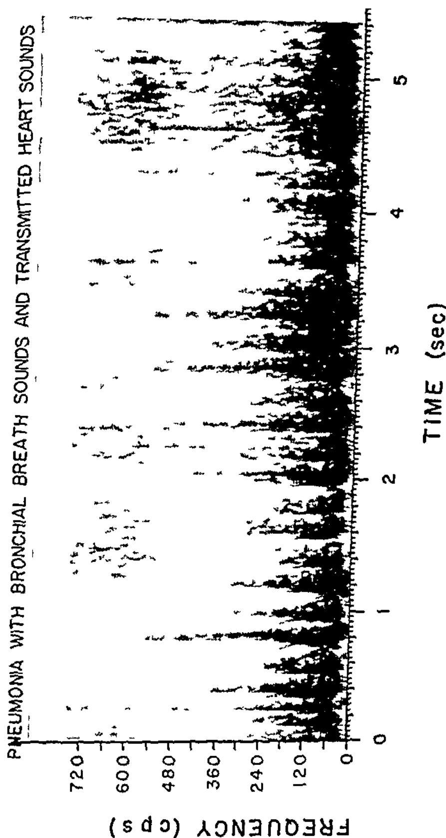


Fig 475 Bronchial breath sounds (at frequency of 400 c p s up) and transmitted heart sounds in case of *pneumonia of the left lower lobe*

to the microphone with abnormal clarity. Pronounced tachycardia was present.

Martini and Mueller (1015) demonstrated in a model that pronounced damping of sound vibration occurs in tubes of 2 to 3 mm in diameter. In tubes of 1 mm in diameter very little conduction occurs. This was the basis for their conclusion that the bronchial breath sounds have their origin in bronchioles larger than 4 mm in diameter. At this level the bronchiole has a cartilaginous wall and its natural frequency would be expected to be little influenced by the presence of surrounding alveolar consolidation. The appearance of bronchial breathing with the development of consolidation is a matter of transmission of vibrations which occur normally. Anatomic studies revealed that in the adult thorax bronchioles of 2 to 3 mm diameter lie 3 to 4 cm from the surface of the chest and in some areas 5 cm. Near the vertebral column in the interscapular area, these smaller airways may be as close as 1 to 2 cm to the surface. These measurements are an indication of the depth of consolidation which is necessary for the development of bronchial breathing.

Using Helmholtz resonators, Martini and Mueller (1015) demonstrated components of approximately 1 000 c p s in bronchial breath sound. In the experience of Cabot and Dodge (208), the highest frequencies lay in the range of 600 to 1 000 c p s.

WHISPERED AND SPOKEN SOUNDS

Corresponding to the presence of bronchovesicular breath sounds the *whispered voice sounds* may be exaggerated in intensity and frequency span, as is illustrated in Figure 476.

When bronchial breath sounds are present the whispered sounds (903) take on the character which gives the phenomenon the name whispered

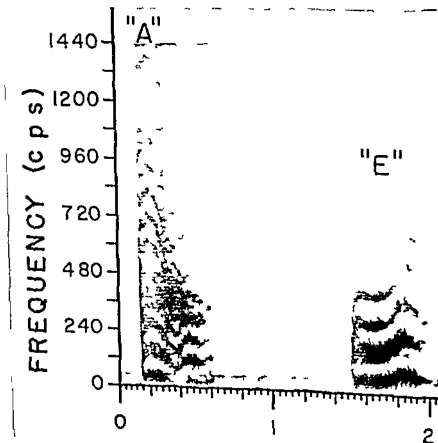


Fig. 478. Spectrograms of sound "E" and "A" spoken directly into microphone

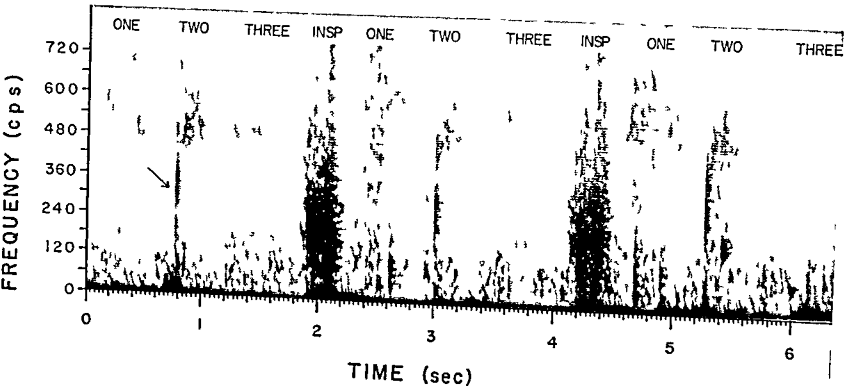


Fig 477 Whispered pectoriloquy changes paralleling the development of bronchial breath sounds in pneumonia Note that the sounds have the same general frequency range as the breath sounds in bronchial breath sounds (475) Note also the sharp t sound of two (a so called plosive) indicated by the arrow

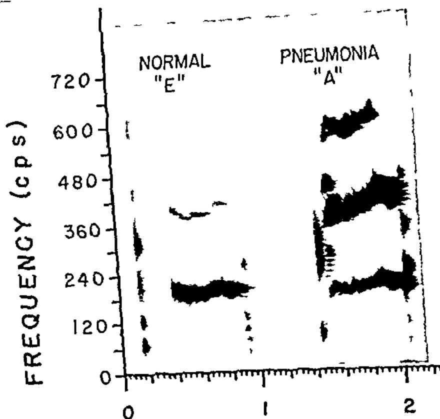


Fig 480 E to A change over consolidated lung

is er an (one two three) The word for one was pronounced E in his province hence the origin of the E to A sign

Froeschel and Stockert (496) found that one vowel sound might be transformed into another with no particular predictability

The patient from whom the recording analyzed in Figure 481 was made had a pleural effusion on the right The alteration is somewhat *different* from that seen in Figures 479 and 480 Here A when detected at the upper level of the fluid sounded to the ear something like ă (as in cat) with a bleating quality typical of egophony The physical changes produced on the side of the fluid are (1) marked attenuation of the fundamental (at 160 c p s) (2) reinforcement of the second harmonic (at 320 c p s) and (3) attenuation of the fourth harmonic (at 640 c p s) The behavior of the hemithorax which is partially filled with fluid is that of a stiffer pass band filter it has a higher center frequency and narrower frequency pass band

In the past much of speculative nature has been written about the physical origin of egophony Norris and Landis (1147) surveyed the theories in this manner

(a) Flattening of noncartilaginous bronchi by pressure of the effusion so that they act like reeds (Laennec) (b) vibrations of the walls of small bronchi by actual collision—interruption of air currents (Wintrich) (c) the articulation of overtones—only the higher harmonics passing through the effusion (Stone) (d) vibrations passing through a thin layer of fluid

Obviously studies by sound spectrography lend support principally to the theory of Stone

Dunn and colleagues (388) studied the acoustic properties of the normal and abnormal chest using a phonation method essentially identical to that discussed in this section The intensity of *elements* in various frequency bands at various

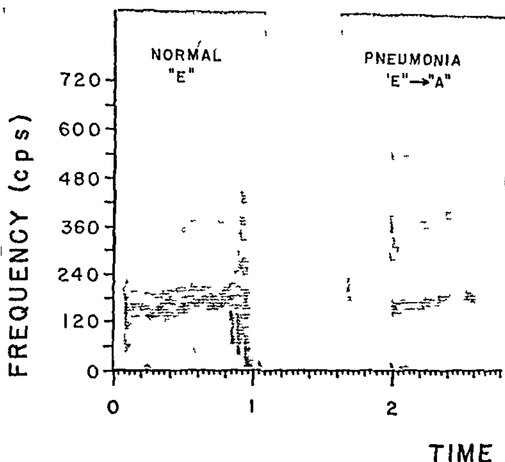


FIG. 479. "E" to "A" change over lung consolidated by pneumonia.

pectoriloquy (Fig. 477). The sounds are high pitched with the same general frequency range as the inspiratory and expiratory sounds in bronchial breathing. (Different cases are illustrated in Figures 475 and 477.) The genesis and significance of bronchial breath sounds and whispered pectoriloquy are identical. It is of interest to note in Figure 477 that the sharp "t" sound of "two" (a so-called "plosive") is evident each time as a circumscribed sound indicated in the first instance by an arrow.

Analysis of the alterations in the *spoken vowel sounds* has yielded some of the most interesting results of the present studies. Contrary to what at first thought might seem to be the case, "E" is, on the average, a lower pitched sound than "A." The analyses of the sounds "A" and "E" presented in Figure 478 were made from recordings of the sounds spoken directly into the microphone. Both sounds have conspicuous harmonics with characteristic curvature. In sound "A" the frequency span is greater, and the most intense (loudest) harmonics are in the vicinity of 360 c.p.s. as compared with 150 c.p.s. in the sound "E."

Over consolidated lung tissue "E" spoken by the patient is likely to sound like "A"—the so-called "E to A" change. The analyses displayed in Figures 479 and 480 demonstrate that this change is due to selective reinforcement (or at least selective increase in transmission) of certain ones of the higher harmonics of "E" and attenuation of lower ones. The result is a sound in which the harmonic pattern resembles more that of "A" than that of the parent sound "E."

This type of physical sign was probably discovered simultaneously about 1922 by Shibley in China (385) and Froeschels and Stockert in Vienna (496). Shibley reported that the five vowel sounds "A, E, I, O, U" all became "A" or "Ah" in a localized area over fluid or consolidation and indeed also over a large goiter. A medical missionary in China, he came upon this sign *rather accidentally* from having his Chinese patients

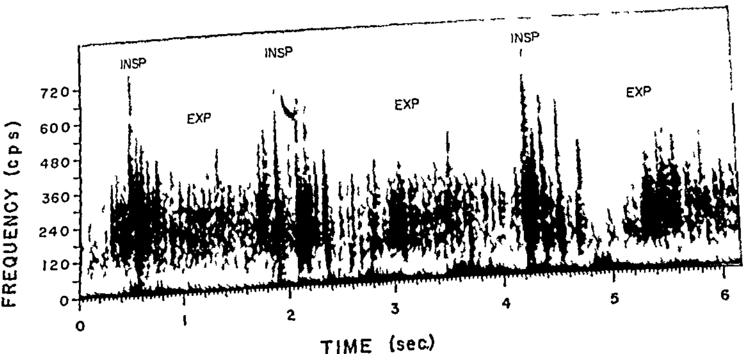


Fig. 482. Moist rales

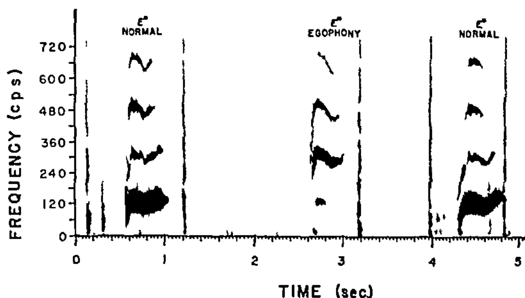


Fig 481 Alteration in E at upper level of pleural effusion E sounded like A and had the bleating quality of egophony

loci was determined As might be expected the method gave results which were the same as those reported here However obviously the method was much more arduous and its final product less graphic

RALES

Rales with the exception of the musical sibilant *ones produced in the bronchioles and* occurring most dramatically in bronchial asthma, are circumscribed noises of limited duration but considerable frequency span It is possible by the method of sound spectrography to provide a physical definition for the extensive descriptive terminology for rales Two extreme examples are illustrated in Figures 482 and 483

The patient illustrated in Figure 482 had extensive bronchiectasis with superimposed pneumonitis Numerous moist, bubbling rales are present in both inspiration and expiration The breath sounds are bronchovesicular in character In the second '*inspiration*' a *wing shaped* harmonic at approximately 600 c p s indicates a slight bronchiolar squeak which occurred at that point Most of the rales in this instance display a fine harmonic pattern

By way of contrast, the patient illustrated in Figure 483 had dry crackling rales limited to expiration Pulmonary tuberculosis was present with predominant involvement of the right apex, from which region this recording was made The harsh character of the inspiratory sound is evident As to the rales these sounds are characterized by a more diffuse uninterrupted frequency *distribution* than *in the moist rales* illustrated in Figure 483 From other experience it is known that sharp sounds with this pattern are to the ear snapping and crackling in quality In studying cardiovascular sound for example it is found that systolic clicks and the opening snap" of mitral stenosis have this same general appearance in the spectrogram

The musicality of the rales in *bronchial asthma* is the result of the presence of conspicuous harmonics (see Fig 484 also Fig 41B) Expiration is prolonged and occupied by harmonics as demonstrated in the illustrations Close scrutiny of the *tracings reveals, as might be expected that more* than one musical instrument" has been recorded One produces a fundamental at 240 and a second harmonic at 480 cycles per second This same sound generator also produces a fainter sound in

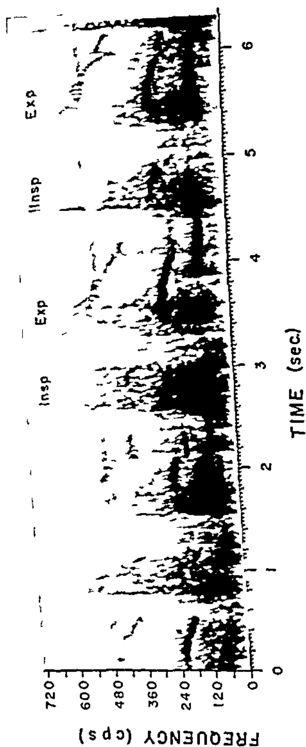


FIG. 181. Musical rales of bronchial asthma. Expiration is much prolonged.

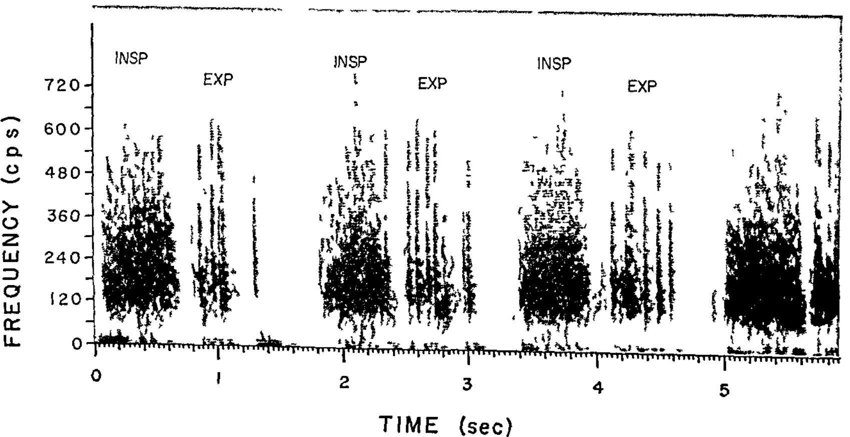


FIG 483 Dry crackling râles in expiration Note the difficulty in differentiating dry râles from pleural friction rub (cf Fig 485)

SECTION VIII

Miscellaneous Sonic Phenomena of Medical Interest

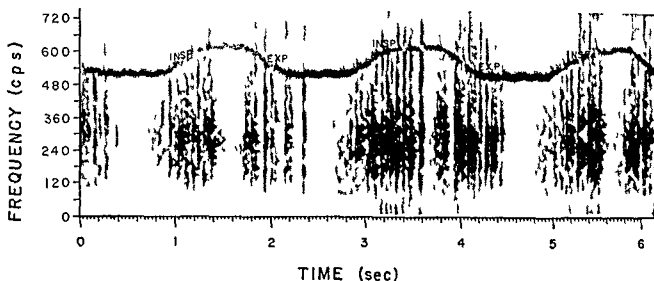


Fig 485 Pleural friction rub in J R (176906) 49 year old patient who demonstrated a pleural rub of several weeks following an automobile accident with rib fractures The rub consists of a series of transients in rapid succession

inspiration In two cycles, late in expiration a pair of harmonics are seen at 180 and 360 c p s The two harmonics crossing at 480 c p s almost certainly were generated in different bronchioles

PLEURAL FRICTION RUB

The mechanism of the pleural friction rub is "stick and slip" (p 138) as is that of the pericardial friction rub At one extreme stick and slip may be so evident that discrete sounds, transients, are evident to the ear and are demonstrated in the sound spectrogram (Fig 485) This coarse type of pleural friction rub may be difficult to differentiate from coarse, dry rales At the other extreme the pleural friction rub may be difficult to distinguish from abnormal breath sounds specifically the exaggerated breath sounds of partial pulmonary consolidation (This is a situation analogous to the confusion of some pericardial frictions for endocardial murmurs) In such instances stick and slip probably is operating independently in innumerably local zones Although in each area a faint transient is produced, in the aggregate these result in a murmur like sound

CHAPTER 25

Miscellaneous Sonic Phenomena of Medical Interest

BOWEL SOUNDS The experienced abdominal surgeon is able to extract much information about intestinal motility or obstruction from the character of the bowel sound. Several authors beginning at least with Walter B. Cannon in 1905 ([illegible]) have studied abdominal sounds (890-965). Some (453) have used these sounds for an objective measure of the effects of antispasmodic drugs. A difficulty of this method is introduced by the fact that the stomach and colon dominate in the production of noises, overshadowing the small intestine which is most sensitive to antispasmodic drugs. The teaching and possibly the research value of a systematic review of the subject of bowel sounds has not yet been explored.

The study of *joint sounds* is in a similarly undeveloped state.

Lian and Odinet (933) observed that in the presence of ascites percussion in one iliac fossa and auscultation (or recording) in the other resulted in the detection of a double sound as opposed to the single sound produced in the normal.

Peltier (1198B) describes a sound conduction test for fracture of the femur and dislocation of the hip. The method would appear to have particular usefulness under circumstances of emergency. With the patient supine, a stethoscope is placed firmly on the symphysis pubis and each patella is struck lightly with a finger. A clear, distinct sound is transmitted by the unbroken bone column on the normal side, whereas a softer, less distinct sound is heard from the injured side. The progress of healing can be neatly followed by means of this test. Bone cysts and tumors and possibly effusion of the hip joint may produce change. Modification of the test should be applicable to fractures in other bones.

SECTION IX

Technical Appendix

TECHNICAL APPENDIX

Prepared with the Assistance of Mr George N Webb

SPECTRAL PHOTOCARDIOGRAPHY

Discussion of the problems of instrumentation for spectral phonocardiography can be divided into the following major parts first the detection of the sound on the chest wall second the storage of these sounds along with the correlative physiological data third analysis of the sound for tonal quality and fourth the final written record (1514)

The equipment must be considered still in the developmental stage Various parts of it are being used daily in clinical studies while the development advances All basic operational functions discussed have been tested and used but one unified easily operated let alone commercially feasible instrument has not yet evolved

DETECTION OF CV SOUND Most heart sound transducers can be placed in one of three classes (1) displacement where no contact is made to the chest wall (e g Groom's microphone (610)) (2) pressure where an air chamber between the chest wall and a diaphragm converts the wall displacement to a pressure (most phonocardiography is done with transducers of this type) and (3) contact where the chest wall drives a sensing element directly See p 503–504 for a more detailed discussion of microphones

Respiratory variations are usually eliminated by having the transducer ride the gross movement of the chest wall with breathing The low frequency ballistic components artifacts for purposes of phonocardiography are attenuated by acoustic filtration inherent in the physical design of the transducers and or by electrical filtering It is important that the undesirable low frequency pressures present at the microphone position do not overload the system and thus generate new high frequency components which have no existence in physiologic fact

Several studies (1031) have indicated that the amplitude of the frequency components of a given heart sound decreases toward the higher frequencies at the rate of about 16 db per octave The ear at low levels of pressure approaching the threshold of audibility, has a sensitivity curve which rises at the rate of about 12 db per octave In many respects this feature of the auditory mechanism is ideal for auscultation The 16-db per-octave slope of the heart sounds suggests that a phonocardiographic system which has a rising sensitivity of about 12 db per octave may be desirable When the full tonal quality of the sound is to be illustrated, low frequency attenuation should be used to the extent that the highest and lowest amplitudes at the extreme frequency limits come within the amplitude dynamic range of the final write-out Care must be exercised in this regard since during the heart cycle the dynamic range may be great at any given frequency level This range again is beyond the scope of the write out and can be best handled by logarithmic amplification after analysis

INFORMATION STORAGE For displaying the full tonal quality of the sound some form of write-out representing many frequency components is required Either multiple simultaneously operating analyzers—such as Mannheimer (1031) Maass and Weber (1001) and others use—are required or alternatively storage and repeated analysis of the same sound We have used the second method which for the information provided seems to have the economic advantage at the present time

All data are first recorded on magnetic tape Selected portions are transferred to magnetic loop or magnetic drum for repeated playback and analysis

The equipment first used to establish the

TECHNICAL APPENDIX

Prepared with the Assistance of Mr. George N. Webb

SPECTRAL PHONOCARDIOGRAPHY

Discussion of the problem of instrumentation for spectral phonocardiography can be divided into the following major parts: first, the detection of the sound on the chest wall; second, the storage of these sounds along with the correlative physiological data; third, analysis of the sound for tonal quality; and fourth, the final written record (1014).

The equipment must be considered still in the developmental stage. Various parts of it are being used daily in clinical studies while the development advances. All basic operational functions discussed have been tested and used, but one unified, easily operated, let alone commercially feasible instrument has not yet evolved.

DETECTION OF CV SOUND. Most heart sound transducers can be placed in one of three classes: (1) displacement, where no contact is made to the chest wall (e.g., Groom's microphone (610)); (2) pressure, where an air chamber between the chest wall and a diaphragm converts the wall displacement to a pressure (most phonocardiography is done with transducers of this type); and (3) contact, where the chest wall drives a sensing element directly. See pp. 503-504 for a more detailed discussion of microphones.

Respiratory variations are usually eliminated by having the transducer ride the gross movement of the chest wall with breathing. The low frequency ballistic components, artifacts for purposes of phonocardiography, are attenuated by acoustic filtration inherent in the physical design of the transducers and/or by electrical filtering. It is important that the undesirable low frequency pressures present at the microphone position do not overload the system and thus generate new high frequency components which have no existence in physiologic fact.

Several studies (1031) have indicated that the amplitude of the frequency components of a given heart sound decreases toward the higher frequencies at the rate of about 16 db per octave. The ear at low levels of pressure approaching the threshold of audibility has a sensitivity curve which rises at the rate of about 12 db per octave. In many respects this feature of the auditory mechanism is ideal for auscultation. The 16-db-per-octave slope of the heart sounds suggests that a phonocardiographic system which has a rising sensitivity of about 12 db per octave may be desirable. When the full tonal quality of the sounds is to be illustrated, low frequency attenuation should be used to the extent that the highest and lowest amplitudes at the extreme frequency limits come within the amplitude dynamic range of the final write-out. Care must be exercised in this regard since during the heart cycle the dynamic range may be great at any given frequency level. This range again is beyond the scope of the write-out and can be best handled by logarithmic amplification after analysis.

INFORMATION STORAGE. For displaying the full tonal quality of the sound, some form of write-out representing many frequency components is required. Either multiple simultaneously operating analyzers—such as Mannheimer (1031), Maass and Weber (1001) and others use—are required or alternatively storage and repeated analysis of the same sounds. We have used the second method which for the information provided seems to have the economic advantage at the present time.

All data are first recorded on magnetic tape. Selected portions are transferred to magnetic loop or magnetic drum for repeated playback and analysis.

The equipment first used to establish the

Fig 486 Circuits for FM modulation of low frequency physiologic data

practicability and utility of this method of analysis included an Ampex 350 stereophonic magnetic recorder, of which one channel was employed for a direct recording of the heart sounds, while respiration and EKG were recorded on the other channel as frequency modulated signals For repeated analysis these two signals were mixed and analyzed by the Vibralyzer of the Kay Electric Company

A newer model uses four channel recording on half inch tape in order that *multiple microphone* recording may be accomplished along with more physiological data

The physiological data—respiration, EKG, and pulse pressures—require a band of frequencies extending down from 100 cycles per second, but no high degree of DC stability Frequency modulation (FM) is required for tape recording of these data Figure 486 shows the basic circuit of the oscillator used for these channels and the heart sound channel when FM is used The first tube is a low gain DC amplifier and driver for the oscillator The second tube is a form of phantastron which does not have DC connection between suppressor and screen It has a linear response of frequency with respect to input voltage for deviation of ±40 per cent of the center frequency

The choice of recording method for the heart sounds requires a comparative evaluation of direct recording and the FM method Direct recording is the method normally used for tape recording of voice and music A high frequency bias is applied at the same time that the tape is magnetized directly by the recorded signal At a given tape speed there is a limit below which distortion becomes excessive on playback because the recorded wave lengths exceed the length of tape covered by the playback head Non uniformity of magnetic material on the tape adds to the distortion In FM a carrier signal saturates the magnetic material, thus minimizing the effect of tape non uniformity Since the information is contained in the frequency of the carrier, its faithful reproduction is dependent upon the uniformity of tape speed In direct recording tape speed uniformity is a second order factor, just as tape uniformity is with frequency modulation The direct recording technique probably provides the higher signal to noise ratio for this work

Although a commercial FM demodulator is used in the analyzer for playback, transistor demodulators are used for monitoring Figure 487 shows the playback amplifier and limiter This is a straight forward transistor amplifier with diode clipping Uniform limiting is achieved over a ten to one change in input amplitude from the playback head The transistor demodulator is a three transistor univibrator consisting of an amplifier stage, common *collector driver, timing* stage, and output filter The one shot univibrator has a period equal to a half cycle of the frequency modulation center frequency As the frequency varies the average height is detected by the low pass filter and reproduces the original modulating wave Again DC stability is not required and the slight drift of this one shot with temperature is

Fig 487 Circuits of playback amplifier and limiter

not important. Most commercial FM demodulators are designed to drive galvanometers, which is not required in this voltage-operating system.

One of the open questions in the final design of a spectral phonocardiograph concerns the best method of repetitively playing back the recorded information for the multiple analysis. The magnetic drum has the disadvantage of a fixed play time and, from our experience, a difficulty in attaining low wow and flutter when an 8-to-1 speed change is required. When the recording heads ride on the drum, excessive wear results, although this may be an acceptable expediency. If the heads are spaced away from the drum, as is common practice in computer work, a very large drum is required to accommodate the frequency response required. For using tape loops, both rotating heads and moving tape with stationary heads have been developed. Whether or not separate transports are to be used and whether all original recordings are to be preserved are considerations to be weighed in choosing a loop playback.

ANALYSIS. The first rudimentary filters for analysis of heart sound were the bell and diaphragm of the stethoscope. These provided a choice between two rather broad band pass filters which aided in focusing on one or another aspect of the sound. When amplitude vs. time recordings (conventional phonocardiograms) were made it was evident that some form of low frequency attenuation was required. Methods vary from a simple 6- or 12-db per-octave attenuation across the entire audible region to very sharp low-cut filters to demonstrate the components above 1,000 cycles (as used by Rodbard (1293)). Another approach was to pass the sounds simultaneously through multiple filters, making multiple amplitudes vs. time recordings. There is obviously an economic limit to the number of such channels.

The analyzing method on which spectral phonocardiography is based was that developed by the Bell Telephone Laboratories (1247, 1248), and called visible speech. In this system, in order to obtain a maximum of information, the abscissa remains time, but the ordinate becomes frequency, whereas intensity of the sound at a

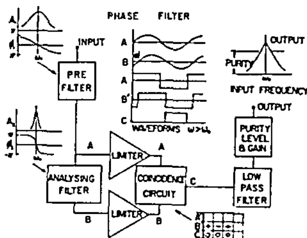


FIG. 488. Schema of phase filter.

given time and frequency becomes a step on the gray scale.

The present method is to scan many times the group of heart sounds to be analyzed. The rate of scanning is eight times the speed at which the original recording was made. This speedup decreases the time required for analysis and allows the use of convenient sizes of filter components.

The analyzing filter operates at a fixed frequency of 15 kilocycles per second. The sounds to be analyzed are heterodyned with a local oscillator so that the lower side band passes through the filter. The frequency of the oscillator is coordinated with whatever mechanism changes the vertical writing rate of the write-out. The output of the filter is used to intensity modulate the write-out.

Two types of filters are presently available. First is a conventional two stage LC tuned filter. As explained on p. 89, the five filter systems we tested are arbitrarily designated A through E. "A" represents a relatively high Q where the filter rings and is quite frequency sensitive with little time information. "E" represents the other extreme where the Q is low and much frequency information is lost. Filter "C" represents a compromise in which some degree of both timing and frequency detail is retained. We have arbitrarily chosen filter "C" as the one having the best timed and tonal characteristics for cardiovascular sound.

Figure 488 shows the basic operation of the second type of filter, the phase filter (722, 1101).

FIG 486 Circuits for FM modulation of low frequency physiologic data

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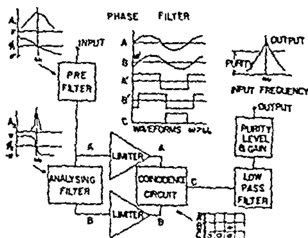


FIG 488 Schema of phase filter

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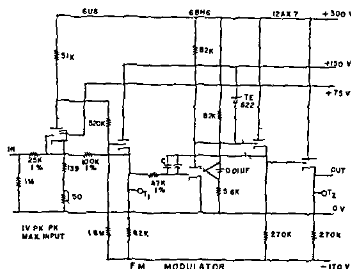


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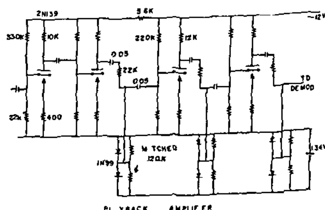


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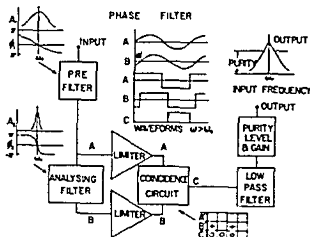


FIG 458 Schema of phase filter

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The purpose of filter A is primarily that of removing the upper side band It provides very little filtering by itself The analyzing filter (A2) is the same filter as that used in the system referred to in the last paragraph However, instead of detecting the amplitudes from the filters the two signals are passed through the two limiters, providing square waves at the points A and B A time difference between the zero crossings of signals A and B exists, depending upon the relationship of the frequency of the input signal to the center frequency of the analyzing filter The coincidence circuit produces an output pulse only for the period that both A and B are negative The low pass filter takes the average value of this coincidence pulse The purity level is a clamp circuit control which in effect removes that portion of the response curve below the corresponding purity level and thus narrows the band width This narrowing of bandwidth has been accomplished without introducing further ringing, such as occurred with the first filter The result of this type of filtering, illustrated in Figure 44, is to accentuate continuous tones over random noise Where the time frequency characteristic of a musical murmur is more important than the actual intensity of the musical tone, the second system is superior

THE WRITE OUT *In the original equipment* a magnetic disk like drum twelve inches in diameter and one half inch wide and a writing cylinder like drum four inches in diameter and eight inches long are mounted on the same axis The electro sensitive paper is wrapped around the writing drum As the drums rotate during analysis a marked stylus moves up the drum changes the frequency of the local oscillator during each rotation and writes the filter output on the record drum The principal disadvantages of this method are lack of precise intensity control in marking the paper a short gray scale range and the ease of smudging the record An attempt to overcome some of these difficulties was made by using photographic paper on a second drum operating on the same axis but in a light tight box above the original drum (Fig 43) The difficulty of manipulating the photosensitive paper under these circumstances shelved this approach

Figure 489 presents a simplified block diagram

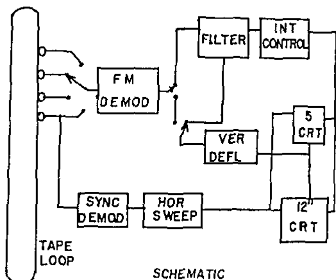


Fig 489 Schema of SPCG system

of what, at the present writing, seems to be the most satisfactory system A cathode ray tube is used for the final display with a camera making a photographic record The unique feature of this system is the ability to combine two dimensional linear deflection pulse intensity information the same as in a TV raster, with the conventional oscillographic display

Either a loop or a drum storage mechanism is used for repetitive playback Two mixed channels may now be used on the drum and four channels on the loop The horizontal sweep of the 12″ monitor tube and the 5″ read out tube is linear and synchronized from the loop Unlike the mechanical system where the magnetic drum and write out mechanism are locked together, the reproducibility here is dependent upon low jitter of the sync pulse uniformity of tape speed, and linearity of sweep The main advantage here is that the analyzed portion can be chosen to be all or part of the loop depending upon sweep speed and sweep delay The vertical deflection of the two C R tubes can be either a linear sweep synchronized with the frequency change of the filter local oscillator or it can be switched to the demodulated signals from one of the recorded channels to provide an oscillogram

The sweep times involved in the raster scan are of the order of 600 milliseconds maximum for the horizontal sweep and five minutes for the vertical during the total analysis The slow sweep

rates place unusual requirements upon the intensity control circuit. The cathode-ray tubes being used are magnetically deflected and electrostatically focused. The beam is gated by the cathode and the grid is used for intensity modulation. For 10 KV accelerating potential and these sweep speeds a bias of 45 volts is required with a modulation signal of 2 volts peak. Since screen burning occurs very soon after the 2 volt level is reached careful limiting circuits are required. The problem of grain lies with the cathode-ray tube and not with the film. What is needed is a homogeneous phosphor.

The relationship between grid drive and light intensity is not linear, so some form of gamma control is necessary. A rooter circuit (11534) is included to compensate for this effect. This is in addition to any logarithmic compression that may be introduced in the filter circuit to compress the great amplitude range of the sound into the film latitude. With the present equipment about 43 db of signal to the rooter circuit carries the beam intensity across the exposure range of the Polaroid film used in making the permanent record.

FUTURE DESIGN CONSIDERATIONS. For widespread use of the spectral phonocardiograph a simplified form of information storage and repetitive playback is desirable for economic reasons. For the sake of reduced size and decreased heat output more use of transistors is indicated. Preliminary design and testing have been made on logarithmic compression, deflection circuit, and power supplies using transistors. For instance the deflection circuits are inefficient although high impedance yokes are used. The cathode-ray tubes using the new transparent phosphor under development by several manufacturers would improve the uniformity and resolution of the final display. More basic research into the adequate display of the tonal characteristics of the impact sounds (heart sounds) is required.

OTHER SELECTED TOPICS IN THE TECHNICAL ASPECTS OF PHONOCARDIOGRAPHY

Microphones

The detection of heart sounds on the chest wall is usually accomplished by allowing the movement of the wall to produce pressure changes in a closed volume (the second general type of method mentioned on p. 499). A pressure transducer is connected to this volume. For the stethoscope the ear is the transducer. The bore, length and elastic properties of the tubing can affect the results obtained by the stethoscope (see pp. 68 to 70). When microphones are used as the pressure transducer a minimum volume of air is introduced between the chest wall and the diaphragm of the transducer; tubing effect can usually be ignored.

For quantitative reproducible results several aspects of heart sound detection need consideration. The pressure which the microphone exerts on the chest wall affects the tension of the surface skin and produces compression of the subcutaneous tissues. As a result sound transmission is affected in a manner which at this time has not been quantified. The variability of these effects between patients is also unexplored. Care must be taken that the large displacement of low frequency sound does not produce pressures in the small enclosed coupling volume of the microphone such as to distort the electrical output. A microphone should be chosen which can either accept the high pressures without distortion or one which has an acoustic filter to reduce their amplitude.

The capacitor microphone depends for its operation on variations of the electrical capacitance between a movable diaphragm and a fixed plate.

One form (610) uses the chest wall or a light aluminum foil on the chest wall as the movable plate. This microphone detects chest wall movement with a minimum of loading of the wall. In addition, since no diaphragm sensitive to air pressure is used, the pick up of ambient room noise is greatly reduced. The sensitivity of this microphone is dependent upon the basic separation of the two plates which should be of the order of a few thousandths of an inch. Although this adjustment presents problems the above mentioned advantages are important. There is no opportunity for acoustic filtering in this unit.

The second form of the capacitor microphone (684) uses a stiff glass diaphragm about the size of a dime. The facts that acoustic filtering can be used, that it can withstand high pressures with

minimum of distortion[1] and that it is made of inert glass, gold, stainless steel and Mycalex recommend this unit. For the purpose of detecting change in the capacitance a charging voltage of about 200 volts is used. The usable signal is a small variation of this charging voltage (of the order of a few thousandths of a volt). High humidity can be a problem because of electrical leakage in the associated amplifying equipment which necessarily have a high impedance.

The crystal microphone has been quite widely used for phonocardiography (1031). In this transducer the movements of a diaphragm transmit a force to a crystal which generates a voltage when stressed. The principle advantage is low cost. The disadvantages are distortion with high acoustic loading, deterioration with prolonged exposure to either high or low humidity, and complete destruction by temperatures over 120° F. Although some (1031) have used this for calibrated work, its chief utility comes in providing a record of the heart sounds in situations in which wave form is not the first consideration and temporal correlation with other types of records, e.g., the ballistocardiogram, is the main requirement.

The *electrodynamic*, or moving coil, *microphone* (88) consists of a coil mounted on a diaphragm and a permanent magnet. The coil is caused to move in the magnetic field by the vibrations of the diaphragm. A complex acoustic filter is re*quired to insure that the voltage output is propor*tional to the pressure on the diaphragm rather than to the velocity of the diaphragm coil combination. In the past the large size and weight of this type of microphone and its susceptibility to pick up of hum has impeded wide acceptance in phonocardiography; however, there are some reports of its use (1033).

Dunn and Rahm (385) described a *pistonphone* which could be used for calibrating microphones. Other uses are suggested by the statement of these workers that it is a "stable and reliable source of pure low frequency tones for general applications in the acoustical fields"; that is, it can be used as a sound generator for studies of sound transmission in the body.

A new type of microphone which may contribute to our understanding *of the meaning of the* heart sounds is the *phono catheter*. This consists of a pressure transducer placed at the end of a catheter to measure the transient pressure conditions inside the heart chambers and the great vessels. Since it is the pressure transients that are of interest it is important that the catheter should not be sensitive to bending and impacts. Careful evaluation of this pick up is needed before the results of its use can be fully accepted. If proved satisfactory, much new *information* can be obtained by this instrument.

Low Frequency Attenuation

As previously stated, attenuation of low frequency signals is necessary in phonocardiography for two reasons. Signals below the audible region should be attenuated sufficiently to cause no overloading in microphone electronics or display in an oscillogram. Because *of the great amplitude range of the heart sounds in the audible region* the low frequency components which have the greatest amplitude need to be attenuated to bring them to a level comparable to the high frequency components which are used for timing purposes.

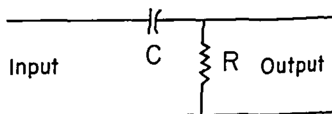


Fig. 490. Circuit for low frequency attenuation

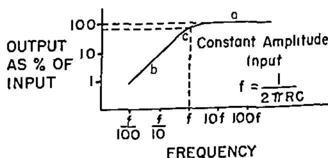


Fig. 491. Output of circuit of type shown in Figure 490

[1] In the condenser microphone there is still risk that strong impacts may overload the microphone and introduce click like sounds which in fact do not exist. The mid systolic click recorded at ILSB in cases of ASD (see Fig. 339) may be a case in point.

(in the case of the heart sound) and for tonal analysis (in the case of murmurs)

Low frequency attenuation in phonocardiography is accomplished by means of a circuit of the type schematized in Figure 490. The electrical circuit characteristic of a capacitor (reactance) is symbolized as X_c and is defined by the formula $\frac{1}{2\pi f C}$ in which f is frequency and C is capacitance. Capacitance is directly proportional to the area of the plates and inversely proportional to the distance between them ($C = K \frac{1}{d}$). Reactance is frequency-dependent. The characteristics of the entire circuit is dependent on the relationship between X_c and the resistance (R). The unitage for both values is ohms. When $X_c \ll R$, i.e., is much less than R (by a factor of 100 or more) then the input about equals the output.

Because of a vectorial relationship between X_c and R there is a phase shift at low frequencies. With an input signal which is of constant intensity over a range of frequency up to 2000 c.p.s., output will match input at the upper end of the frequency scale (line A of Fig. 491) and there will be no phase shift. At low frequencies there will be a linear relationship (line B in Fig. 491) between frequency and output with, however, a 90° phase shift. In this range the attenuation is 6 db per octave or 20 db per decade. At intermediate level of frequency when X has a value between 10R and R/10 there is a curvilinear relationship (line C in Fig. 491) this being with respect both to amplitude of response and to phase shift

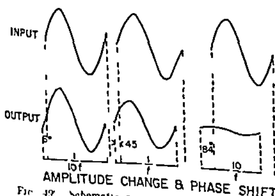


Fig. 492. Schematic representation of phase shift accompanying low frequency attenuation by circuit of type diagrammed in Figure 490.

the transition zone between the two areas of linear response. See Figure 492 for a schematic representation of phase shift and amplitude attenuation.

Acoustic attenuation of low frequency components of sound as was practiced by Einthoven (Fig. 1b) and many since him has the same characteristics as those indicated in Figures 491 and 492 and the circuit represented in Figure 490 is the electric analogy of simple acoustic attenuation.

It is unlikely that the inevitable phase shift which accompanies low frequency attenuation introduces serious error into phonocardiography. Any errors are in the timing of cardiac events and it is unlikely that these will exceed the 0.01 sec. limit which is the order of exactitude in

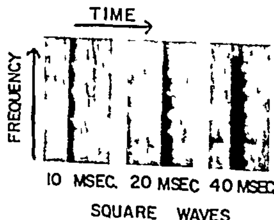


Fig. 493. Artifactual splitting of square wave.

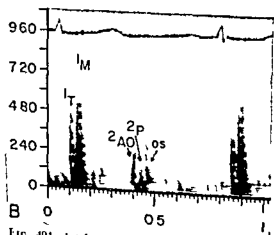


Fig. 494. Artifactual splitting of tricuspid closure sound (T) and mitral closure sound (M) when sounds at apex in mitral stenosis stretched out on time scale and played into spectrograph with high gain.

minimum of distortion[1] *and that it is made of* inert glass, gold, stainless steel and Mycalex recommend this unit For the purpose of detecting change in the capacitance a charging voltage of *about 200 volts* is used The usable signal is a small variation of this charging voltage (of the order of a few thousandths of a volt) High humidity can be a problem because of electrical leakage in the associated amplifying equipment which necessarily have a high impedance

The crystal microphone has been quite widely used for phonocardiography (1031) In this transducer the movements of a diaphragm transmit a force to a crystal which generates a voltage when stressed The principle advantage is low cost The disadvantages are distortion with high acoustic loading deterioration with prolonged exposure to either high or low humidity, and complete destruction by temperatures over 120° F Although some (1031) have used this for calibrated work, its chief utility comes in providing a record of the heart sounds in situations in which wave form is not the first consideration and temporal correlation with other types of records e g , the ballistocardiogram, is the main requirement

The *electrodynamic* or moving coil *microphone* (88) consists of a coil mounted on a diaphragm and a permanent magnet The coil is caused to move in the magnetic field by the vibrations of the diaphragm A complex acoustic filter is required to insure that the voltage output is proportional to the pressure on the diaphragm rather than to the velocity of the diaphragm coil combination In the past the large size and weight of this type of microphone and its susceptibility to pick up of hum has impeded wide acceptance in phonocardiography however there are some reports of its use (1033)

Dunn and Rahm (385) described a *pistonphone* which could be used for calibrating microphones Other uses are suggested by the statement of these workers that it is a stable and reliable source of pure low frequency tones for general applications in the acoustical fields ', that is, it can be used as a sound generator for studies of sound transmission in the body

A new type of microphone which may contribute to our understanding of the meaning of the heart sounds is the *phono catheter* This consists of a pressure transducer placed at the end of a catheter to measure the transient pressure conditions inside the heart chambers and the great vessels Since it is the pressure transient that are of interest it is important that the catheter should not be sensitive to bending and impacts Careful evaluation of this pick up is needed before the results of its use can be fully accepted If proved satisfactory much new information can be obtained by this instrument

Low Frequency Attenuation

As previously stated, attenuation of low frequency signals is necessary in phonocardiography for two reasons Signals below the audible region *should be attenuated sufficiently to cause no* overloading in microphone, electronic or display in an oscillogram Because of the great amplitude range of the heart sounds in the audible region the low frequency component which have the greatest amplitude need to be attenuated to bring them to a level comparable to the high frequency *components* which are used for timing purpose

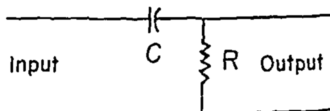


FIG 490 Circuit for low frequency attenuation

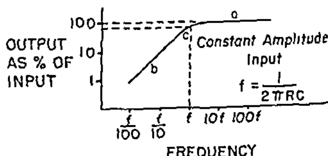


FIG 491 Output of circuit of type shown in Figure 490

[1] In the condenser microphone there is still risk that strong impacts may overload the microphone and introduce click like sounds which in fact do not exist The mid systolic click recorded at HSB in case of ASD (see Fig 339) may be a case in point

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phonocardiographic mensuration. Particularly is this true since the rapid deflections of higher frequency composition (above 100 c.p.s.) are, as a rule, used for making temporal measurements.

Storage of Heart Sound Information

If one is to record on magnetic tape for subsequent analysis both oscillographically and spectrographically, it is desirable to have only as much low frequency attenuation as is necessary in order for the tape to accommodate the wide dynamic (i.e., intensity) range involved. Thereafter, the little altered signal can be put into the spectrograph (which can accommodate a wide intensity range) and the tapes can be used for teaching purposes; the necessary attenuation for low frequency and high frequency (Leatham), stethoscopic and logarithmic (Rappaport), oscillograms can be imposed between the tape and the oscillograph.

Artifacts of Overloading

A square wave imposed on the spectrograph, is displayed as indicated in Figure 493. A transient *with frequency content over the entire range* encompassed by the instrument occurs both with onset (A) and offset (B) of the wave. If the square wave is of short duration an apparently unitary transient results. If a low frequency vibration is recorded on the tape at excessive gain there may be a cut off of its top so that in effect something closely approaching a square wave is produced. The spectrogram might be interpreted as representing a split sound. This appears to be what happened when the sounds of the Hufnagel valve were recorded (1077). The initial recording on tape was made in the operating room soon after placement of the valve in the aorta and with the microphone held by the surgeon close to the prosthesis. The spectrogram shows splitting of both the sound of opening and that of closing. The conclusion was arrived at earlier (1077) that there is asynchronous impact of the ball on the corners which hold the valve open in cardiac systole and that in closing the ball impinges first on one side of its circular seat and then on the rest. It is almost certain that the tape was "overloaded" in the initial recording and that artifactual splitting resulted by the mechanism described above. No such splitting is observed when the Hufnagel sounds are recorded from the surface of the chest without overloading. We have also observed artifactual splitting on this same basis when the valve closure sounds are recorded at very high gain. Figure 494 presents such a case: both the tricuspid closure sound and the mitral closure sound appear to be slightly split. *In this case the sounds were re-recorded at high gain in* order to overcome the foreshortening of the *frequency scale which was an inevitable accompaniment* of spreading the time scale by the method then available.

Several times earlier (p. 160, footnote p. 501) reference has been made to the risks of introducing artifactual high frequency components when the *phonocardiographic system is overloaded* by intense low frequency components at any stage from the microphone to the final display. Ljunggren (959) points out that this can happen with filter systems such as used in the Mannheimer-Stordal method. Rodbard (1293) uses a sharp low cut filter to eliminate components below 1000 c.p.s. He then displays high frequency "spikes" which he interprets as evidence that cardiovascular sounds (both heart sounds and murmurs) are basically a series of closely spaced transients. Although an extensive critique of the method and results is not possible, one would suspect that the "spikes" are high frequency artifacts generated by "hitting" the microphone or the filter excessively hard with low frequency components.

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